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ENJOY BETTER BREEDING

Lyn Tower

To my Grandson

Brian Towd

Merry Christmas 1988

& love from

Nana

(Lyn Towd)

88

in a
wise,

Enjoy Better Breeding

ILLUSTRATED by Gail Harries and Leanne Reely

ISBN 0-7316-3507-8

THIS BOOK IS DEDICATED TO

MY GRANDCHILDREN

Paul, Seran, Brian, Roy, Kristian and Taryn TOWER.
Leanne and Renee PEARSON.

If any of you grow to share my interest in animal breeding, I hope my book will be of value to you.

A VERY SPECIAL THANKYOU TO:–

My husband Stan, for his encouragement and support, and for his tolerance of the time spent on the book – as long as he didn't have to miss a meal!

My son Lindsay (Ty), for reading the manuscript and for the many useful suggestions which have improved the presentation. Especially as this chore fell at the end of the financial year, and his busiest time.

My daughter Carole, who said I couldn't write a book without her name in it . . . . but because she shares my love of writing and word usage has, in fact, been of great help.

Monica Stewart, Sarina Qld., who initially lit my fire of interest in hereditary factors. Without her unstinting counsel over the past 15 years, this book might never have been written.

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Leanne Reely, Qld., for help with the kennels and domestic chores, and for drawing most of the diagrams.

Seran Tower for drawing the hearts – which grandma found tiresome.

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NOTE: Because this is a step by step learning book, seeking information via an index could mislead the reader.

Most words you seek will be found in the "Glossary" at the end of the book. For ease of reference, the glossary gives the chief relating chapters.

For a better understanding of the use of a word, that chapter should be read in its entirety.

AUTHOR'S FOREWORD

This book is written for the learner breeder. I have assumed that the reader is totally unaware of even the basics of selective breeding. I have written it with the idea firmly in mind that some readers may not even have studied biology during school days. Some may not have had any tertiary education at all.

When we take up breeding – whether it is dogs, cats, goats, horses or any other animal, we need useful information which we can apply to our own particular interest.

We need simple explanations. We need to proceed slowly step by step, and at this early learning stage we need as few complicated issues as possible.

For example, we might like to know what colour we are likely to produce when we mate a pure black dog to a pure white one. Or how colours are inherited in cats. Or whether a long coat fault can be reduced in a breed which stipulates only short coat is acceptable . . . or vice versa.

We are mere breeders trying to grasp some basics of breeding, either for our own interests and improvements – or with the intention of studying the subject in depth once we've found an easier route into the maze of technical jargon found in most existing text books.

This book will help you to do this. You will learn about some modes of inheritance. This means that when you plan a mating you have a bit more chance of working out for yourself how some existing qualities might be retained, while other undesirable faults may be avoided.

I make no claim to any qualification in the field of science. I am merely a dog and cat breeder. With my husband I have bred a few cattle, sheep and pigs in the past. Breeding for the livestock market where one's income depends on quality – as well as quantity, makes a breeder get down to serious study. In doing this I personally struck great difficulty in grasping the basics of hereditary factors from existing books written by the experts in this field.

I have, therefore, merely attempted in this book to explain some useful basics in more simple terms than usual. I am fully aware that in some instances the treatment is unorthodox. Any omissions of detail, or deviation from accepted genetical terms is my conscious effort to simplify explanations.

I hope these pages will offer you useful guidance. My book will help you to proceed to a more advanced study if you so desire.

This book is devoted to "modes of inheritance." All mention of nutrition, hygiene, housing and common ailments etc. has been deliberately omitted. Nevertheless, the reader will understand that time should also be devoted to these subjects. All are of equal importance to responsible breeders.

DURI N. S. W. Evelyn Rebecca (Lyn) TOWER
Australia 1988

ONE

PREAMBLE

A large part of my life is spent on the telephone attempting hasty answers to involved questions on animal breeding. I then find it necessary to follow up the phone calls with lengthy letters. Logically it seems to be more practical to put what help I can offer into book form.

I have spent most of my life associated with the German Shepherd Dog – and more recently the Himalayan Cat. Alongside my husband I have *dabbled* in cattle, pigs and a few other farm animals.

My aim in writing this book is to show readers that even a very elementary knowledge of basic inheritance factors can be of absorbing interest and practical help to ALL animal breeders – whatever animal interests you.

Some people learn quickly – other more slowly. In this book I have assumed that, like me, the reader may be an "average learner." Therefore, if I could learn these basics I feel sure you can also do so. However, I also admit to being a book worm, and a very tenacious character – for me it was a challenge! If you approach it with the same determination I am sure you will succeed.

For those who prefer to whistle and let fortune be their guide, I have this story to tell.

Our best producing milking cow gave birth to twin heifers. Not having bred cattle before we clapped our hands – not one more milking cow but two! "Not so!" the experienced neighbour farmer told us – been in cattle since he was a boy and what he didn't know wasn't worth knowing (his words!).

He said we would have to sell the twin heifers on the meat market because twin calves, he believed, are infertile. In the

meantime we'd bought a good pure bred bull. "Damned good bull that" the *expert* informed us many months later staring at our twin heifers – both heavy in calf. What he didn't know was that for twin calves to be infertile one must be a male – in which case the bull calf takes all the breeding genes from his sister and leaves her infertile (explained in more detail in Chapter 13).

Because of this farmer's ignorance we might well have parted with two valuable milk producing cows.

This is a good example of the cost of ignorance. Learning at least the basics of genetics is highly desirable to any breeder of any species of animal, bird fish or fowl – or anything at all! Especially if learning the basics will help us to avoid many of the pitfalls we may otherwise encounter – and it will! Even so, we must remain aware that it will not solve all problems.

Whenever you select a male for your female you are dabbling in genetics. Even if you erect a barrier against learning any genetics, you are still utilising the genes those animals are carrying when you mate them together. In other words you CANNOT ignore the laws of nature because the laws of nature will persist in spite of you. Therefore any information we can learn in this regard is going to be an asset to improving stock, and may save a lot of initial mistakes we may have made without that knowledge.

I'm always amazed at how easily the public accept the term "BREEDER" as depicting someone very knowledgeable in the animal world. The world is crammed full of *breeders* . . . and it is truly amazing how few really do know much about the material they work with.

I'm sure that many breeders believe the tooth fairy really does exist, and that wishes will be granted overnight. With one show win under their belt, overnight they become so called "experts." They purchase one animal of a breed (good, bad or indifferent) and when mating time comes around they are masters of their craft. One successful working animal and they are convinced its offspring will all be clones of the parent.

Ah! If only it were all that easy! It is not!, As most enthusiasts eventually discover. But by the time they do discover it they have already dumped on the market many poor and mediocre stock which they need not have produced with just that bit more thought and study beforehand.

We have only to hang around a show ring for a couple of hours to know that breeders in general can be very misguided about the material they work with. Let me quote some examples. I'm sure

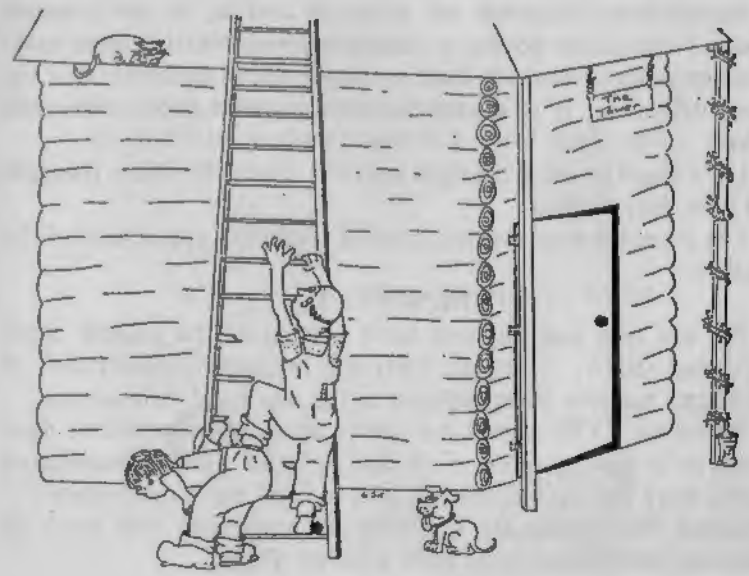
you've all heard a breeder blaming a male for every fault in the book. This breeder will blame somebody else's animal he has used at stud, for every fault he produced in the offspring.

He'll even blame the male for the number of offspring (which mostly depends on the number of eggs the female sheds.) Some breeders can be heard blaming the female parent for the amount of males in a litter which in fact the male controls. Others complain about the amount of long coats they get in a litter when they wanted short coats – which they obviously did not understand the inheritance of.

The mechanism of inheritance is not simple – and I would not like to suggest it is, or can be! It is indeed very complicated, and at times so complex that sometimes the experts cannot agree. One said that it is a difficult subject to simplify without distortion, and in writing this book I'd be the first to agree.

However, it IS going to be kept simple. Even the experts had to begin somewhere and everyone begins by learning the simple basics which is the purpose of this book. Getting a foot on the ladder of success means starting at the bottom.

So let's get down to work.



Two

Phenotype/Genotype

Breeders often plan matings on the exterior appearance the animals involved . . . i.e. how the breeder's eye sees the potential parents.

We call this **TYPE** breeding. We may not know anything at all about the likely genetical inheritance of the animals involved. This kind of mating generally depends solely on the merits of the outward (physical) appearance of those parents.

We may also plan such mating on the show wins of one or both parents . . . or the glamour of "import" behind the name of one or both animals. A mating may also be planned on the working merits of animals – or on the requirements for the commercial market.

Irrespective, what we are doing is looking at the physical (external) merits and hoping to duplicate them. What we hope to do is produce more of Mum or Dad, or maybe the best of both. Our big dream, of course, is to create dramatic improvements over both parents.

Let's begin by using the right words – especially where the right word is so easy to learn.

The accepted word for this physical (external) appearance of the animal is:–

PHENOTYPE

We can now use this one word instead of the lengthy three words used above . . . i.e. the "physical (external) appearance" of the animals has now been reduced to the one word "phenotype."

When we TYPE mated our above animals we relied on their phenotype to give us more of similar, or better quality. Sometimes this will work for us, but often it fails to fulfil our expectations . . . sometimes the results are positively disheartening and leave us wondering how things could have gone so wrong.

Perhaps if we'd put a bit more thought and study into the other half of the animal, i.e. the genes it may be likely to carry, we may not have wasted time on this mating in the first place.

If both animals carry favourable genes for the qualities a breeder requires, the breeder may hope for the improvements dreamt about. If the animals carry no favourable genes for improvement then that mating cannot give them.

This genetical side of the animal is called its:–

GENOTYPE

Just this one word instead of lengthy descriptions.

PHENOTYPE . . . the animal we see before us.
GENOTYPE . . . the hidden genetical make up of the animal.

Another word we might learn in this chapter is PROGENY – it simply means the offspring the animals produce. So the father and mother's progeny are their sons and daughters.

An example of the difference between phenotype and genotype is the erect ears which are required in some animals, while drop ears is an undesirable fault.

We can see in our parents that in phenotype they both have erect ears, and get quite a shock when from these two erect eared animals out pop a few drop eared progeny. In other words we did not know these parents were carrying drop ear genes until we mated them. Of course, once the fault shows up we are then made fully aware of the problem carried on the genes – even though the phenotype did not show such fault.

The question then being how might we go about getting rid of it again, or at least reducing the incidence of it?

A hit and miss method could be a sheer waste of our precious time.

A more realistic approach would be to learn the *mode of inheritance* of any particular undesirable fault — and use that knowledge to our benefit.

If we have some idea of how a problem is inherited in the first place we are part way to solving it. Also if we know how a desirable character is passed on we have a better chance of obtaining it.

It is not easy to fully understand how ALL faults are inherited. It is very important here to be sure you understand that not ALL traits are inherited in the same way.

However, in animal breeding many of the traits (desirable and undesirable) *ARE* inherited in a simple manner. This being so, the reader will greatly benefit from the simple explanations in following chapters.

Readers who have reached this far will agree that this chapter has been extremely easy to follow. Further study will also be explained step by step, and kept simple.

In addition each explanation will be repeated over and over — and there will be plenty of opportunities for revision.

In learning genetics there are so many difficult words which grimly guard the approaches to understanding. There are terms which have to be used if you are to proceed to other text books on completion of this book. Where such words are unavoidable I have attempted explanation of the word prior to the use of it.

All this will surely encourage you to proceed. However, as one politician said "Life wasn't meant to be easy" — therefore some degree of effort, and concentration, on your part is essential.

THREE

DOMINANT/RECESSIVE — An Easy Start

Gregor Johann Mendel lived in the years 1822-1884. He was a teacher of science in Austria, as well as being a monk in a local monastery. He discovered the DOMINANT and RECESSIVE **mode of inheritance.** Also he is credited with what we term "Mendel's law of segregation" — which you will be learning about in following chapters.

Mendel worked with varieties of peas, but countless experiments have since proved that what he found with garden peas applies equally as well to animals, fish, fowl, mice, flies, man, and even to bacteria and fungi.

The garden peas he worked with were coloured red and white. He found that red was DOMINANT and white was RECESSIVE. So we'll begin by making sure we fully understand these two words.

We talk of a dominant person — a person who takes the lead and over-rides other people. We might say this person uses his weight and his influence to CONTROL other people. This type of person does not wipe out his weaker acquaintances . . . he dominates them!

So dominant genes can be likened to the dominant person. They dominate over others and take control. They mask other genes. We have to use the word "mask" because the dominant gene does not wipe out a recessive gene, it merely masks it — hides it! Dominant therefore, merely means the stronger gene — the one able to mask, hide and over-ride a recessive.

The opposite to dominant is recessive. Recessive might be termed the weaker genes which allow themselves to be over-ridden, masked, hidden by the dominant gene.

Simplified these can be remembered as:—

DOMINANT: the power to mask (put into hiding) a weaker character. Dominant can be remembered as the strong one overpowering the weaker one.

RECESSIVE: is the opposite. It is possible for a dominant gene to mask this recessive gene and put it into hiding. Recessive can be remembered as the weaker one. It is important to remember that it doesn't get wiped out — merely masked!

Let's have a look at Mendel's red and white peas. What he began with was a *Pure* red and a *Pure* white. The important word here is *PURE*. The red had no gene for white. The white had no gene for red.

The red inherited the red genes from BOTH its parents. One red gene from Dad and one red from Mum. Ditto the white. It is important to understand that genes have NO effect in SINGLE form (the exceptions to this rule are too involved for this book). One gene of whatever colour is inherited from Dad and the other gene of whatever colour is inherited from Mum.

This meeting of one gene from each parent ensures that a colour carried by those parents is passed on to the next generation.

So the red had to get one gene for red from Dad and one gene for red from Mum, and the white also received one gene for the colour white from each parent.

For simplicity we can term the red "R" and the white "w". Because they have to receive one from BOTH parents the red got an R from its Dad and an R from its Mum — making it RR. The white the same so it becomes ww.

In genetics the dominant is shown in capitals and the recessive in lower case. Just by looking at the way it is written we know that RR is dominant and ww recessive. In Mendels experiments he found that nature determined that the red colour was dominant to the white . . . RR was dominant to ww.

PURE RED	RR	Pure white	ww
(DOMINANT)		(recessive)	

Remembering that these parents received one gene for that colour from Dad and one from Mum, it also makes sense that in turn they also pass down ONLY one of that gene to each offspring.

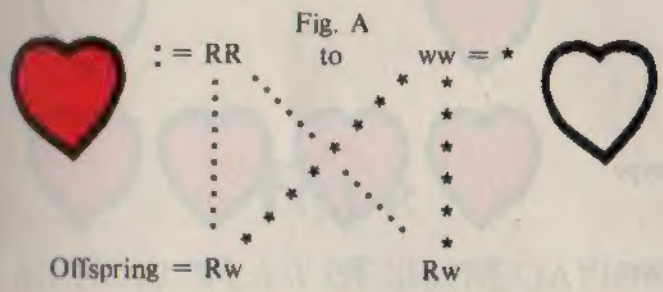
Dad passes down one R to each offspring.

He does *NOT* have any other colour to pass!

Mum passes down one w to each.

She does *NOT* have any other colour to pass!

So we can pretend to do a mating of these two colours, RR RED to ww white, and we can then see what Mendel found.

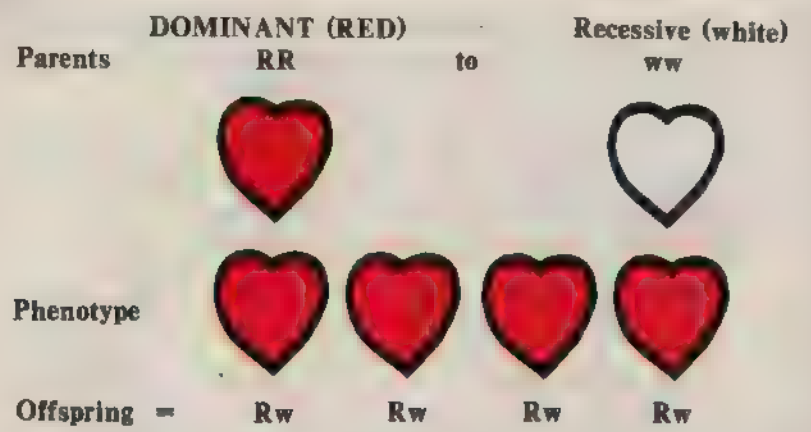


This clearly shows that the offspring are not genetically the same as the parents. The two offspring shown have received an R from Dad and w from Mum and both now carry the genes for **RED** and **white** — they are now Rw. This fact does not alter no matter how many offspring there are from this mating — they ALL receive R from Dad and w from Mum.

If this mating were dogs, cats, pigs etc. where there are multiples of offspring in one litter, the above facts would still remain the same — each pup, kitten, piglet etc. could receive only one of the pair carried by each parent (one from each parent). Each offspring would therefore carry one dominant gene and one recessive. Remember again that Dad couldn't give w because he didn't have it in the first place to give. Mum couldn't give R for the same reasons. So they each gave one of what they did have to each offspring. In this case R from Dad and w from Mum = Rw.

Where the parents were *PURE* for their colour, the offspring are not. The offspring are a mixture of the two colours we began with . . . they are ***IMPURE.***

Unfortunately we cannot see this is the specimens we produced, because Red being DOMINANT has **masked** the white recessive. All these offspring will be phenotype the DOMINANT colour — in this case RED. But all are carrying the white in recessive where it is hidden and we won't see it until we do another mating, which brings the masked white out again — which is explained in more detail later. For now we are still working on the mating of the pure parents.



This first generation are the dominant colour Red (phenotype red) . . . . because white is recessive!

But ALL are white carriers

The clue here is to remember ALL are phenotype Red — seen by us as Red, with absolutely no white showing at all. But all are carriers of white — so the "phenotype is *DIFFERENT* from the genotype."

To show this fact more clearly I am going to use a white spot on the red hearts for the carriers — this will be used throughout the book. It is for diagrammatic purposes only and has no other significance.

Therefore our above mating has produced all Red coloured offspring, carrying white so they are:—

So if we mate pure red to pure white, the dominant will overpower the recessive and the resultant offspring will be red in colour, but ALL carrying the hidden (unseen) white — and we will go on to see what happens when we mate the impure offspring.

This is all quite simple and straightforward. It will all be repeated in different forms in future chapters, and will all fall into place as you go.

FOUR

MENDEL'S LAW OF SEGREGATION

In our mating of RED to white we produced impure offspring carrying both colours — but seen by us as *RED*. (Phenotype red). We established that the red colour was the dominant colour. The white colour had disappeared altogether in that first generation, but was hiding away there in each and every offspring . . . . without exception! . . . . . 100%. Also established was that in carrying genes for both colours, this first generation are termed impure for either colour.

However, the *most important lesson in this chapter* is that THIS HIDDEN RECESSIVE WHITE WILL COME OUT AGAIN when we do the next mating of the impure progeny, i.e. in the second generation. This is called . . . .

"Mendel's Law of Segregation"

In any breeding this is one of the most important basics to learn for future improvements. But before delving any deeper I have to explain some relevant genetic abbreviations.

The PARENT GENERATIONS ARE CALLED:—

P1 — 1st parent generation . . . parents
P2 — 2nd parent generation . . . grandparents.
P3 — 3rd parent generation . . . great grandparents.

This obviously refers to the parents involved in our mating, and to the ancestors behind those parents. It merely makes our reference to them easy and precise.

The OFFSPRING GENERATIONS ARE CALLED:—

F1 — first offspring generation: First filial
F2 — second offspring generation: Second filial
F3 — third offspring generation: Third filial

The letter 'F' means FILIAL and simply means offspring — a son or daughter etc. . . It springs from the word FILIATE — in relation to our study this means to bear a child.

The term F1 is commonly used to describe the first generation from two breeds within a species. For example, the first generation offspring of a cross between one breed of cattle and another — or between one breed of dog and another. In rural areas this term is often used in advertising cattle, sheep or pig sales. It clarifies the cross breeding of what is offered for sale, without lengthy explanations.

Most of us know what the word HYBRID means when it comes to discussing flowers and plants. The hybrid is the result of the first cross between two different PURE members of a species. (F1 — first filial. First generation). In relation to our study here it is used to clarify the first cross of two different pure colours.

Our impure offspring carrying both RED and white are our F1 generation. Our hybrids. We are about to mate this F1 generation. We will then understand Mendel's Law of Segregation . . . .

Remember we have mated our pure P1's (RR to ww) and produced impure F1 offspring (Rw). It is the impure F1's which we are now going to mate together.

Dominant in CAPITALS — recessive in lower case.

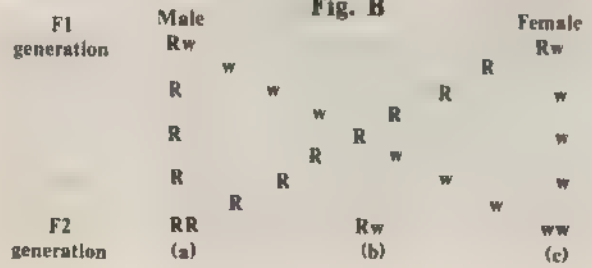
For simplicity in Fig. B below I have shown only three offspring . . . and connected together only (a) PURE RED and (c) pure white . . . (b) has inherited both colours.

This clearly shows the theory involved and to be sure you understand this, you might like to draw some connecting lines of your own for (b) — which is the Rw impure offspring.

The white spot in the red heart represents impurity — i.e. red but carrying white recessive. It is used for diagrammatic purposes only and has no other significance.

If you do not understand it at the first study, do not quit — remember it will be repeated over and over again and will fall into place as you go.

Fig. B



The F2 generation produces:—

Phenotype: **2 red** **1 white**

Genotype: **1 pure red (a)** **1 impure red (b)** **1 white (c)**

and explained in detail later

Homozygous = 2 RR and ww (alike) (pure)

Heterozygous = 1 Rw (unalike) (impure)

(a) Male passed R to one offspring and female has an R to pass. Because they passed it to the same one this offspring will now be RR — *PURE* for RED . . . seen by us as RED.

(b) Male passed R to another, white Mum passed w = Rw — and then we have another impure like the parents. Red to our eyes, but carrying white in recessive. In other words the white cannot be seen in the animal carrying it . . . but it quite positively is in there — in recessive!

(c) Male also passed his w to another offspring, white the female also passed her w.

NOTE: This is where our white came out again. This re-emerging of the white is of extreme importance to us — in particular where such feature is an undesirable one . . . in which case it can be seen and discarded. Alternatively it can be used to advantage if it is a desirable one.

Remember ww was PURE for white! and it has to be seen by us as white because it has no red in it. Remember that if this offspring carried R it would be red — not white!

So now we have the *three* different GENOTYPES in the one litter (the word litter used for clarity).

We have (a) RR (b) Rw (c) ww

(A) RR – red colour and PURE for red

(B) Rw – red colour but impure . . . with recessive white

(C) ww – white colour and PURE for white.

Note: Two red coloured offspring and one white.

In a later chapter we can use some diagrams to mate these three different genotypes with each other. So if you haven't yet quite grasped the principles involved you will be taken through it again in a different form.

However, by now I think you will have gathered that knowing this law of segregation – even though my explanations are very rudimentary – you can use it to great advantage in your own breeding establishment.

(i) It means that the physical appearance of F1 offspring is no indication of its genotype. But its gentoype is most likely to emerge into sight in the second generation . . . when segregation takes place according to Mendel's Law.

(ii) It means that the unwanted features of the poor, or mediocre animal used previously for breeding, are most likely to emerge again in the second generation . . . when segregation occurs! This fact can either take your breeding project back to square one, or advance it.

The statement made above requires explanation! The second generation tends to reveal the material you are working with. If it brings poor qualities to light, you either bury your head in the sand and ignore the warning signs – or alternatively you can use what you learned from it to launch into a more enlightened future.

On the other hand, the F2's may show you many of the desirable features you required. In which case you can then advance your immediate breeding improvements accordingly . . . . and you may reap the rewards at a much faster rate.

(iii) It means that if a crossbred animal was used previously for breeding, while the F1's may appear to be pure bred the proof of their mixed ancestry will emerge when segregation occurs in the F2 generation. This allows you to plan accordingly.

Such knowledge can be used to considerable advantage in any breeding establishment – no matter what you breed.

Before leaving this important chapter there are two new words to learn.

They are used in Fig. B on pages 18 and 19.

	HOMOZYGOUS	**and**	**HETEROZYGOUS**
Pronounced.....	Homo zy gus		Het ro zy gus
and meaning	pure – or alike		impure – or unalike

They look very big words and sound nasty enough to scare anybody off. Like all big words they only need the meanings explained.

HOMOZYGOUS can be remembered as meaning like to like. Homosexual is one sex to the same sex, and we all know what the word homosexual means. Homo – like to like: the same type! Pure . . . therefore

Homozygous means a gene pair the two members of which are similar. As in the case of (a) above . . . RR two genes similar – in this case red! Pure for red! Also in the case of (c) above . . . ww and in this case white! Pure for white.

HETEROZYGOUS is the opposite. It can be remembered as unalike. Male to female is heterosexual. Different sexes. Disimilar types Hetero – unalike. Impure . . . therefore

Heterozygous means the members of a disimilar gene pair. As in the case of (b) above . . . . Rw two disimilar genes . . . not pure for either red or white.

If you have any difficulties at this stage in learning these two words, use the words PURE and IMPURE and go back and study these words when you become more familiar with the principles of the simple dominant and recessive mode of inheritance.

FIVE

ACCEPTABLE/UNACCEPTABLE QUALITIES

Let's take a breather from the study. I'll use this chapter to have a look at some of the more interesting aspects which understanding the basics opens up to us. Perhaps it will give you the incentive to proceed through the ensuing work.

Every breed of animal (within every species) has an accepted STANDARD. Within this standard some things are favoured and others are frowned on. For example a long coat is acceptable in an Old English Sheepdog — but frowned on in a German Shepherd. The Landrace pig is expected to have flat ears — the Large White erect ears. The Persian cat a long coat — the Siamese a short.

These features are controlled by the simple Mendelian factors you are learning from this book. It is simply a matter of learning what features are acceptable and which are frowned on. Then learning which is dominant and which is recessive.

Many recessives are undesirable but there are exceptions. For example a recessive long coat gene in the German Shepherd Dog is undesirable. In the Himalayan cat the recessive colour points are desirable. In the Dobermann Dog the black is dominant to brown, but the brown is not undesirable.

So you can see how important it is that you know the faults and virtues of what you breed — also what is acceptable and what is not, and most importantly, whether or not such characters are dominant or recessive.

In most examples in this book the recessive genes are shown as the undesirable. It is important that you realise why I have done this. It has been shown this way purely for simplicity. Not all recessive genes are undesirable . . . although most are!

Sometimes the recessive is the type preferred by breeders, as in the example of the colour points of the Himalayan cat.

In livestock sometimes the recessive is the type preferred by the market. We prefer white flesh on our chickens, but I'm told the U.S.A. and other countries prefer the yellow flesh. Because white flesh is dominant to the yellow in this case, the task of overseas breeders to produce yellow becomes easier than it is for Australia, or U.K. etc to produce the white. In a small group discussion five breeders out of six thought this would have to be the other way around — i.e. they considered it would be easier to breed the white because the white is DOMINANT. This is not so, as you will discover when we have done a little more study.

Recessives breed true. Yellow flesh in poultry is *PURE* recessive and as such carries no white flesh gene. Therefore U.S.A.'s desire for yellow flesh is easy to produce because yellow flesh birds put to yellow flesh birds will breed only yellow flesh birds. The phenotype shows us the genotype.

Whereas white flesh birds, white being dominant to yellow, can carry the recessive for yellow flesh which will continue to give both white and the undesirable yellow flesh disliked by U.K. and Australia. The problem associated with the white is that the phenotype does not show us the genotype, and yellow has to be constantly discarded from the stock. This is all quite easily understood. If you are not sure about this from the previous chapters, you should be by the end of this book.

Many dominant/recessive characters have already been well tested and proven in the past. Therefore, it is generally no great hassle to find out if any trait is inherited in simple dominant/recessive manner. Nor for that matter inherited in any other manner. Until recently there was not a lot of research done into dogs and cats — but plenty into mice, rabbits, cattle, pigs etc. Nowadays most of the information you require regarding the inheritance of certain characters is readily available via geneticists working on a variety of animals. Many have also written books on the mode of inheritance of the majority of characters.

For example, a glance through a few available books will tell you that . . . .

In cattle black coat is dominant to red and white.
The white face of the Hereford is dominant to plain.
In cattle uniform colour is dominant to spotted.
Black in the Dobermann dog is dominant to brown.
Black coat in horses is dominant to chestnut.

White fleece in sheep is dominant to black — with a couple of exceptions.

In pigs small size is dominant to large — and long faces to short "dished" faces. White is dominant to all colours.

In German Shepherds the short coat is dominant to long coat.

In Himalayan cats the short coat is dominant to the long — and the self colour dominant to the colour points.

. . . . and I could go on and on and on!

The main benefit from knowing which traits are dominant and which are recessive is that the traits can then be controlled to some extent within a breeding programme.

This will not guarantee overnight success, but it will enable you to seek out the best qualities and reject the defectives.

This means that what you do keep for breeding is then getting closer to the ideal with each subsequent mating. This does not mean that you will eventually breed perfect specimens — there is not enough time in any one breeder's lifetime for this even if it were possible . . . and it is not! We deal in live creatures and no matter who you are, or how carefully you plan, that little man in the sky is going to have the last say. And from time to time he can come up with the most weird and wonderful surprises.

Nevertheless there are many unwanted or undesirable traits that can be controlled when you understand how these traits were inherited in the first place.

It would be remiss of me not to point out the danger in believing all genes to be simple dominant/recessive pairs — or that the answers to reducing the incidence of problems are that simple!

Even so, knowledge of the inheritance of dominant and recessive characters does solve a great many of the irritating problems which confront breeders.

So let's get back to learning a little bit more about it.

SIX

RATIO OF INHERITANCE

Back to our study, with a recap on what we've already covered.

We mated pure red to pure white. RR to ww. This produced impure offspring in the first generation.

Let's repeat that paragraph in correct terms.

We put homozygous red RR to homozygous white ww and produced heterozygous Rw progeny in our F1's.

Then we mated our impure progeny together and the picture changed into one red, another red carrying white, and one white.

Let's put that into correct terms.

We put heterozygous Rw to heterozygous Rw and our progeny were one homozygous RR, one heterozygous Rw and one homozygous ww.

One pure red. One impure red, One pure white.

By showing you that the basics are not all that difficult after all, perhaps we've knocked a few bricks off that barrier you previously put up against the subject. Now we can take this a bit further.

The RATIO of inheritance is extremely important to our study, because it is a CONSTANT ratio. So if we just alter the above a little I can explain this ratio without confusing you.

Let's say we've mated our Rw to Rw and we had FOUR offspring instead of the three which were used for simplicity.

Our four offspring would then be

RR Rw Rw ww

One pure red — Two impure red — One pure white

1 — 2 — 1

25% — 50% — 25%

The ratio of inheritance = 1 : 2 : 1

In other words 25% pure red. (homozygous)
50% impure (heterozygous)
25% pure white (homozygous)

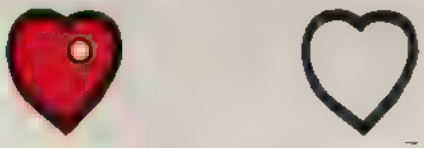
Mendel proved that this ratio of 1 : 2 : 1 or 25% : 50% : 25% holds good ALL the time. But not necessarily all in one litter . . . i.e. it is taken over multiples of matings, and multiples of offspring. In other words you may not get this exact ratio in a one-off litter. But knowing that this ratio has been proved constant means that the significance of it, as well as the outcome of it, can be put into practical use.

If we assume that white is an unwanted fault then the white above would have to be culled. If you use an unwanted recessive you increase your incidence of it. Alternatively, if white is a desirable feature you would wish to use it to increase your incidence of it. Genetics and the ratio of inheritance determines this for you.

Let's see what happens when we say "I know it is a fault, but maybe I'll be lucky and not get it again so I'll use it anyway".

We are going to mate our Rw to our faulty ww.

Impure red = Rw to Pure white = ww



Rw to ww

and without any diagram (but Fig. C following shows this) we can plainly see that we have increased our unwanted white. We now have three w's and only one R.

We have increased the undesirable from 25% to 50% and decreased our desired red from 75% to 50% — this is in comparison with our Rw to Rw mating which produced only 25% pure white.

Just in case you haven't got that clear let's do it again in a different form.

Our previous mating Rw to Rw had two dominant genes for red and two recessive genes for white.

The progeny were 3 reds and one white = 75% red : 25% white.

Just putting on one side the fact that a couple of our reds are not pure (which I'll return to later) the facts are that in phenotype 75% were the desired red and only 25% were the unwanted white.

Also in the original mating we had at least one desirable pure red offspring to base our future plans on, plus a couple impure reds to speculate with.

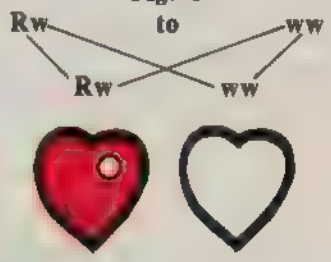
By using our faulty specimen for the Rw x ww we now have NO PURE red at all. We've also increased our gene bank for white.

Remember previously you learned that BOTH parents had to carry it for it to manifest itself — and you also learned that a single gene has no partner so cannot come out.

Without even looking back at the previous work you can see that Rw to ww cannot give you a pure red offspring. Between the two parents there are not two R's to pass down. This mating can only give impure red and pure white.

Therefore by using the faulty specimen not only will you certainly see it again, but you've actually doubled your chances of doing so. Yes! I said, and meant "doubled". In diagram form (Fig. C below) you may see more clearly why this is so.

Fig. C



GENOTYPE: 2 Offspring. 50% impure red 50% pure white.

Multiple offspring? Two or twenty offspring, the ratio of 50% impure red and 50% pure white will still apply.

From this it can plainly be seen that we have:—

(i) No pure red at all.

(ii) Impure red have decreased from 75% to 50%
(iii) Pure white has increased from 25% to 50%.

By using the undesirable colour for this mating we actually took a backward step away from the improvement we originally sought. Even worse is that we took a mighty big deliberate step towards the problem we wished to avoid.

The principle remains the same whatever we breed. The clue to it all is to *KNOW* what your desirable or undesirable features are and then to *KNOW* whether they are dominant or recessive.

Going back to the white and yellow flesh of poultry in the previous chapter. The abbreviations for the above mating would be shown in our simple form as Wy to yy. If it was your wish to breed yellow flesh it is the recessive y you are breeding towards and Wy to yy would take you *CLOSER* to the colour you want. Once you get your breeding to a *PURE* recessive, which means yy to yy you will then produce *ALL* yellow flesh . . . no white at all!

If you wished to breed white flesh this mating of Wy to yy would be taking you *FURTHER AWAY* from your goal. To breed towards more white you would need more capital W's . . . i.e. WW to Wy . . . then your incidence of white flesh would increase while the yellow would decrease. Once you get your breeding to a *PURE* dominant, which will mean WW to WW you will then produce *ALL* white flesh.

Breeding the double recessive is easy because the phenotype shows you its genotype. In the above example because its flesh is yellow this tells you it is *PURE* recessive yellow and will breed true.

Unfortunately this is not so with the dominant. The white fleshed bird may be white *PURE* or it may be white *IMPURE* and its phenotype will not reveal which one – these have to be mated again to prove themselves in the F2's when the yellow (if there) will come out again. If you do not want yellow, it would be wise to discard this from further breeding, because it can only take you back to square one.

SEVEN

DOMINANT/RECESSIVE – PUT TO GOOD USE!

Most readers by now will clearly understand this simple Mendelian mode of inheritance. However, before moving on let's just briefly re-cap and then proceed a little further. This will enable us to see how an understanding of this can be put to practical use.

Dominant is shown in capitals – recessive in lower case. To make study easy so far, I used the initials R for RED and w for white.

The correct term is R for Red and a lower case r for the recessive – i.e. Rr . . . or B for dominant BLACK and b for the recessive i.e. Bb.

You take your first capital letter from the dominant gene and its recessive partner is given the lower case of that letter.

In the white/black pigs the dominant was the white – so this would be written down as Ww. W for the dominant white and w for the recessive member of this pair of genes.

Such abbreviations are accepted and used by geneticists. They avoid misunderstandings and save lengthy explanations.

A mere glance at these abbreviations and they know exactly the dominant of that particular pair of genes.

For breeders' purposes it does not really matter if our abbreviations are not always those coined by geneticists – the main thing is to understand the underlying theory behind the abbreviations. Our concern is to learn as much as we can absorb, and to put what we've learned into practical use.

Anything we can learn, and in particular anything we can use in our breeding plans, has got to be far better than working in ignorance.

Therefore, to aid comprehension in this regard and to abbreviate the records you will wish to keep, coin your own abbreviations if it will help you.

Short coat dominant to long coat could be Ss, Erect ears dominant to drop ears Ee.

If curly coat in your breed is dominant to straight you might use Cc.

As you read further into this book you will realise how important it is for you to consider such genes generally working in pairs — not singly! For you to work with them in pairs you need to understand the above. If you don't do so, the text books you will want to delve into when you've completed this book, will still be a maze of technical jargon . . . because there are variations to this which are too complex for our purposes.

One way of explaining the importance of genes generally working in pairs is to remember the following:—

THE WHITE PEA HAD A PAIR OF WHITE GENES. TO BE WHITE IT HAD TO RECEIVE A WHITE GENE IN DOUBLE DOSE . . . . A PAIR! — ONE FROM EACH PARENT!

THE RED PEA HAD AT LEAST ONE RED GENE. THE OTHER BEING RED OR WHITE. TO BE RED IT EITHER HAD TO RECEIVE TWO RED GENES IN DOUBLE DOSE . . A PAIR! — ONE FROM EACH PARENT. OR ONE RED AND ONE WHITE — ONE FROM EACH PARENT.

Remember also that if red had been present in the white pea it could not have been white. The dominant partner of the white was red, therefore if it had a red gene at all it would have to be showing us red! If you have not yet grasped this you should now return to chapters three and four, and recap on dominant/recessive and the pure/impure studies.

In the pair of genes involved with the pure red, or the pure white, you might say that either one was the colour it was because the other colour was absent. So let's think about this absence aspect.

Red was absent from the PURE white — white was absent from the PURE red.

I know that this sounds a bit like saying "I'm not there because I'm here" — or that somebody is sane because they aren't crazy. However, it does seem to work out in animals that very often the problem facing us is not in what is present, but in what is absent. A pure white is often the absence of black pigment — for example albino. The liver (brown) colour in dogs is generally the absence of black pigment.

This absence of something is likely to be carried on the other half of a gene pair, i.e. it is being masked, or it may not exist at all — and it can be very much more complicated than depicted here, but the value of this book must be in its simplicity.

To me, the importance of this is very relevant. If we consider a problem which we have to reduce (or eliminate) very often we see only what is before us and overlook the fact that such problem may not be ■ much the presence of it — but the absence of something else.

Knowing this often means that some problems can be reduced much faster in a breeding establishment. It also means that time is not wasted in trying to produce a feature which doesn't exist in that animal's genotype in the first place.

For example if black is dominant to brown (liver) in your breed, the mating of two browns will not produce a black for you. The black dominant is ABSENT. In this instance a breeder would be wasting precious time and energy trying to breed black from brown parents. A black parent would have to be introduced after which ALL F1 progeny would be black, carrying brown — i.e. Bb, as shown in Fig. A. . . F2's would then segregate the ratio into 1 : 2 : 1 — as explained ■ Chapter Six.

This "liver" colour cropped up in German Shepherds some years ago. Somehow the colour began to be termed "brown". In terming it a brown gene the inheritance factors of the colour got sadly misunderstood. In fact, this colour was absolutely no threat to the breed in general nor to any serious minded breeder, because the genetics did not lie with the colour "brown" but in the absence of black pigment. All it required to keep it in recessive was the re-introduction of black pigmentated animals — preferably homozygous BB. In other words it was a simple Mendelian dominant/recessive inheritance. And as such, control was as simple ■ child's play.

By studying the content of this book, any German Shepherd breeder who still produces liver, or does not know the mode of inheritance of liver, should now be able to put this into perspective. With the remedy so easy to follow, and apply, the cure is simple to achieve.

I hope that when you've finished my book you, the reader, will have a much more informed approach. Especially towards unwanted traits inherited in this simple manner, which are relatively easy to reduce.

As I mentioned before, it really makes no difference what you breed – dogs, cats, goats or anything else – the simple Mendelian dominant/recessive mode of inheritance remains the same.

So we can now outline the main points again.

To get us into the subject in an easier way we used w for white instead of r, and we began by assuming our P1's were homozygous red RR and homozygous white rr. This time let's do it in a more orthodox manner.

Fig. D

We then mated our red impure Rr x Rr together and found that the white came out again, where we could see it and recognise it for what it really was.

F1 mated together Rr to Rr
produced
F2 result RR Rr Rr rr

R passed from Dad and R from Mum = RR
R passed from Dad and r from Mum = Rr
r passed from Dad and R from Mum = Rr
r passed from Dad and r from Mum = rr

resulting in F2 *genotype* ratio of:–

RR **Rr** **Rr** **rr**
1 **2** **1**

25% Red **50% Red** **25% white**
Homozygous **Heterozygous** **Homozygous**

and before leaving it let's do it again phenotype

. . . . . AS WE SEE THEM!

RR Rr Rr rr

Phenotype **3 red** **1 white**
75% red **25% white**

However, do remember that while I have shown the solid red to clarify how we see it, we are by now very well aware that the centre two hearts are carriers of recessive white . . . even though we can't see it in the animal before us!

They are shown as Rr.

At a glance, this genetic abbreviation tells us:–

1. One gene is DOMINANT and the other is recessive.
2. So, although the three reds look all one colour to us, the two animals in the centre are in fact IMPURE red.
3. Those two animals in the centre are carriers of the recessive.

EIGHT

SELECTING THE DESIRABLES

When a recessive unwanted trait shows up in the manner described in previous chapters, it actually makes selection a little easier for the breeder.

We can see the problem and cull it out. By eliminating it from our breeding programme we can then concentrate on the animals which we can see are carrying the desirable traits. (However, we must not forget that some of the remaining choices are carriers of that unwanted trait).

This is what I would call taking the cream off the top. It is commonly known as "breeding up". Far sighted breeders adopt this method. Only the best you've bred is good enough for future breeding plans. The poor (unwanted) quality is then systematically discarded as it shows up.

I once wrote an article on the above necessity of discarding the undesirables. A kindly breed lover wrote to the magazine in concern for all those animals so systematically discarded — what happened to the poor wee things? . . where do they all end up?

I think it goes without saying that we are discussing animals discarded from a *BREEDING* project — animals which are in some way or other unsuitable for further breeder, or for our purposes. Many such animals have other uses. For example on the domestic scene they may make wonderful pets and there is absolutely no reason why they cannot be found pet homes — on condition they are de-sexed of course, because let's face the facts — if they are unsuitable for us to breed from then chances are they shouldn't be bred from at all.

Assuming we have a problem of drop ears in an erect eared breed. Also assuming erect is dominant to drop. We have to keep

remembering that whatever the problem, if it stems from a simple Mendelian factor the ratios will always work out the same. Again at the risk of boring those fast learners I have to repeat this in diagram form — but for variety I'll show it in another form which you will find in text books — a form which many people find easier to grasp.

Fig. E

		Male E	Male E (Pure erect)
Female (Pure drop) e		Ee	Ee
Female (Pure drop) e		Ee	Ee

F1 generation all Ee

This represents a cross between pure erect EE and pure drop ee. All four progeny are the same Ee.

We've done the mating of the carrier parents before and we know that in the F2's we got our hidden recessive back again — drop ears in this example. And we learned the ratio genotype and phenotype.

Fig. F

	E	e
E	EE	Ee
e	Ee	ee

F2 generation

In this example three are erect eared and one is drop. Of the three erect, one is pure i.e. carries two erect genes EE. The other two carry the masked drop gene. The fourth is drop eared.

GENOTYPE

EE	Ee	Ee	ee
1	2		1

PHENOTYPE

EE	Ee	Ee	ee
	3		1

Genetically we have 25% pure for erect
50% impure (carriers)
25% pure for drop

Phenotype we have 75% erect
25% drop

If you are now beginning to yawn with the boring repetition EXCELLENT — you've made it!

We can now move on.....

We have four offspring in this litter. And we are assuming we have three erect and one drop.

We are now seeking the cream for future breeding. This seeking of our more correct and sounder stock is for overall purposes and not merely concerned with a show ring. Nevertheless if you are breeding for show points — or commercial qualities — in fact whatever you are breeding for — you would clearly have this selection in mind. Also if you know your breed's requirements you will be considering the debits along with the credits.

On the above principles you may overlook some very minor defects but you cannot afford to bring any major defects back into your breeding establishment.

So our drop eared has to be discarded. Using what you should have discarded won't do you any good in the long run — remember we proved that the use of a defect increases your incidence of it.

We've assumed we have four offspring from the above litter. Three erect eared and one drop.

EE Ee Ee ee

EE is the *cream* of this litter for the future elimination of the ear problem.

Ee are *carriers* but can in fact help us to further reduced the incidence of the fault.

They may also help us to increase the incidence of the cream.

But we have first to prove these — which I'll return to later.

ee is the *defective* unwanted relation.

In breeds which have multiple births these three streams exist most of the time. The idea is:—

(a) keep your cream once you've identified it.

(b) Mate and eliminate again from your intermediates — provided all the other qualities of the animals merit it, and of course it is just as important to know a good specimen when you see one, as it is to understand the Mendelian Law of segregation. This attention to genotype and phenotype absolutely must go hand in hand.

(c) Discard the defective poor relation.

This is how a knowledge of genetics can be put into practical use.

Carriers of a single faulty recessive gene will pass it on to only a percentage of the progeny — carriers of a double recessive will pass it to ALL its progeny — 100% . . . a very good reason for discarding it.

Recessives breed true, just the same as pure dominant breeds true. In simple terms this means that when the genes on each side of a pair of genes are similar, i.e. erect ears EE or drop ears ee, the drop ears will breed just as true as the erect. Drop ears ee is as homozygous (pure) for drop ears as EE is for erect.

If an animal is showing a defect which is considered disqualifying, degenerate or uneconomical in that breed, using it for breeding is only going to breed more of the same for you.

This chapter was written mainly to reinforce this fact. It is crass stupidity to retain an animal actually showing you a pure recessive defect which is a disqualifying, or degenerate problem . . . no matter how good you consider it in other respects! To profess to be a serious breeder or indicate that you breed the best you can for the market, knowing full well you bred from an animal you ought to have discarded is not being honest — with yourself, with your breed, nor with your buyers.

Idealism? No, just plain common sense! Moralizing? No, just hopefully explaining the benefits of understanding the Mendelian law of segregation.

Returning, as promised, to our impure Ee's. These, as we have repeatedly learned, are carrying one E for erect ears and one recessive e for drop. They are heterozygous.

Of course what we have here is a return to our F1 generation previously well explained in the red and white examples. And we've just repeated it again with the erect/drop ears above.

We know by now that the next F2 generation brings out the defect again — discard that, and we have left the three erect eared.

The difficulty lies in the fact that it is impossible to visibly see which of the erect eared animals in the F2's are pure and which are impure.

Until they in turn are mated when out pops the pure drop eared again.

In simpler terms we have 75% phenotype erect eared animals in our second generation. Of these some will be pure and others impure and we can't see the genes they carry so we have to mate them again to bring the pure recessive out again.

It is not possible to cull them if we can't see any physical evidence of the problem.

Therefore, all erect eared could be considered as suitable breeding material provided they are physically and mentally sound and of the correct requirements. Here it has to be stressed that the breeding unit is the entire animal — not an isolated pair of genes. Nevertheless where that isolated pair of genes controls a very serious problem this must also be of paramount importance in any breeding programme.

What then happens is that the one which carries the unwanted gene if mated to another also carrying that gene, will again segregate into the ratio of 1 : 2 : 1 (25% pure erect 50% impure 25% pure drop). We can then again keep our erect and discard the drop.

This will then go on continuously until the gene bank for "erect" ears increases while the gene bank for "drop" decreases. So we are constantly increasing the incidence of pure erect while culling and/or reducing the incidence of drop — within our own breeding establishment.

You might say that rather than taking the cream off the top all the time, you are constantly weeding out the poor. But by doing this you are then taking the cream off the top anyway.

In this way it is possible over a period of time to visibly watch the quality increase, and the problems reduce with every litter — or birth. Certainly it is not going to happen overnight. It is going to take you quite some years.

After all, this is what realistic breeding is all about. In the animal world we certainly do not have tooth fairies who grant wishes overnight.

It is fine to go out and buy from some other breeder in order to get something to brag about if that's your scene — or to buy foundation stock elsewhere which has been selectively bred. But if you expect to be producing similar quality from that purchase in say ten/fifteen years time, then at some stage it will be necessary to learn genotype selection as well as phenotype.

In other words it is wise to learn this system of "breeding up".

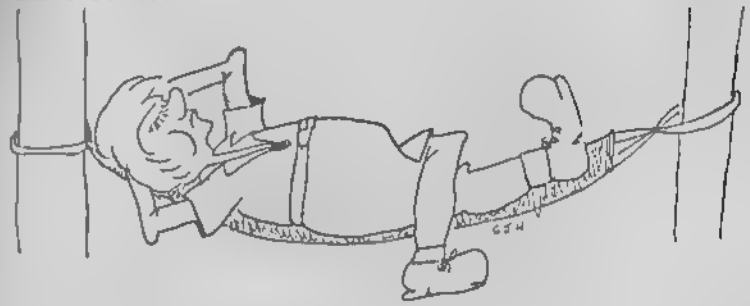
Other wise, your entire breeding effort will consist of depending

on constant purchases from more serious breeders, in order to correct the problems which you create for yourself.

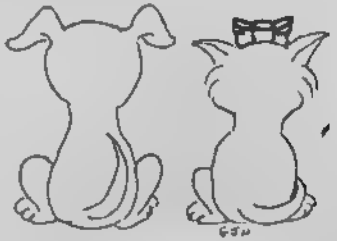
Alternatively you might find yourself continuously cleaning out your entire stock and starting all over again with new stock purchased elsewhere . . . a costly price to pay for lack of learning!

If you are expecting to be breeding successfully for many years to come, you will create many problems by not understanding the modes of inheritance — and you will avoid a very considerable amount by making the effort to learn more about the inheritance factors.

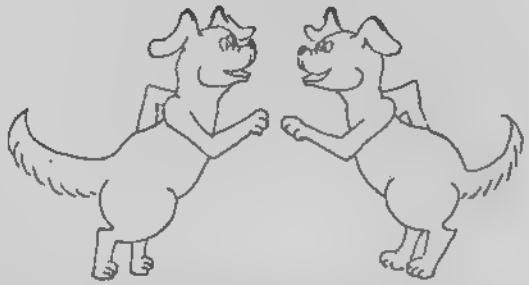
LESS OF THIS



TO BREED MORE ADORABLES



AND LESS ODDITIES.



NINE

DOES LIKE BREED LIKE?

We've all heard the old saying that "like breeds like." Do you believe this is so . . . or might this be a very misguided saying?

Early in the book type mating was discussed, and generally speaking type matings do not take the genetical make up of the animal into account.

In order to fully explain why genotype is just as important to a breeder as phenotype, I have to take our studies into a little more depth — but I will still keep it as simple as possible, and let's see what you think about "like breeding like" when you understand a little more.

Going into colours again — and into black and white will enable me to keep it simple. There are six possible variations we expect to get from the matings involved with the colours black and white. That is to say, matings involved with the DOMINANT B and the recessive b. These six variations remain the same for the erect/drop ears, short/long coat and any other simple Mendelian inheritance.

On the study we've done so far are you able to work these six variations out for yourself? Don't get uptight if you can't. I am going through them for you.

1 BLACK BB to BB BLACK

2 BLACK BB to Bb BLACK CARRIER

3. BLACK BB to bb white

4 BLACK CARRIER Bb to Bb BLACK CARRIER

5. BLACK CARRIER Bb to bb white

6. white bb to bb white

(horses used for diagrammatic purposes only)

If we put No. 2 BB to Bb into diagram form this should simplify it. Only this time we'll do it in a more customary way and introduce a couple of new words.

MEIOSIS explains the dividing of sex cells. It is involved with segregation — the separating and coming together again of chromosomes in the sex cells, which will be explained more fully in a later chapter.

If you are taking your partner out to a big event you do not get up from your chair and leave. You first go into the bathroom to shower, groom and PREPARE for the big event.

Meiosis is the dividing and preparation of the sex cell division to meet its partner for the big event of fertilisation.

GAMETES are half cells formed from the Meiosis division.

You will grasp the meaning of these two words by the end of this chapter.

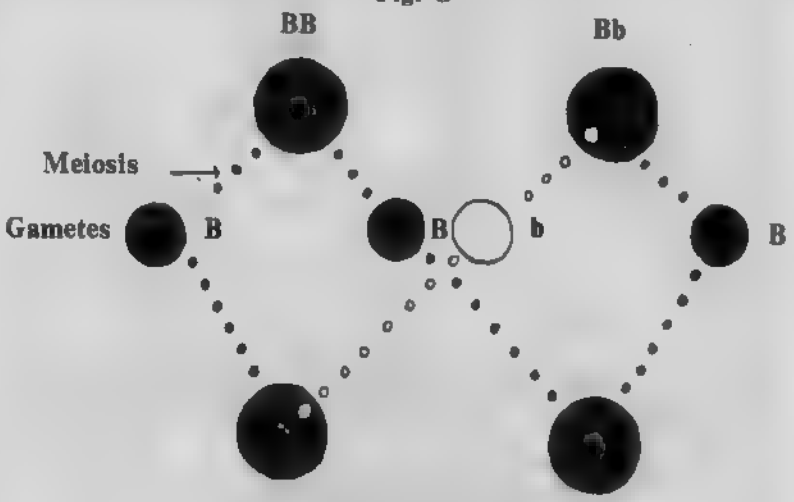
FERTILISATION unites the two halves into a new being with genes inherited from BOTH parents, but which are arranged in different form. The only two individuals being exactly genetically alike are identical twins.

Once again don't get anxious if you can't yet grasp those words — they will fall into place eventually, but I have to use them now to explain my diagram.

Simplified the above merely means that the BB and the Bb above, and in Fig. G, divide (segregate) in Meiosis. For one

dominant B to go to one offspring and the other recessive b to go to another it is obvious they must separate at some point or other. We have to understand how they separate so that we can also know how they come together again (rearrange). We have to know why each new being is individually unique.

Fig. G



In Fig. G the parent on the left is solid black, while the one on the right is carrying white. The carrier passed the **single** white recessive **b** down through the gamete to the offspring on the left — still in single form! This offspring also received **B** from the other parent, so it is a carrier **Bb** like the parent who passed it. The right parent also has the dominant **B** which it passed in single form through the gamete to the offspring on the right. Together with the **B** from the left parent this offspring is **BB** pure black.

In text books you will find this single gene called an ALLELE. One is an allele of the other — the allele **b** is a partner of the allele **B**. The pair usually control contrasted characters, in this case black and white.

When you get all this worked out you can then do similar diagrams for the other five variations above. You should come up with the following and if you don't you'd better try again:—

1.	**Genotype**	**BB x BB**	**= 100% BB**			
	Phenotype	**black black**	**All black**			
2.	**Genotype**	**BB x Bb**	**= 50% BB**	**50% Bb**		
	Phenotype	**black black**	**All black**			
3.	**Genotype**	**BB x bb**	**= 100% Bb**			
	Phenotype	**black white**	**All black**			
4.	**Genotype**	**Bb x Bb**	**= 25% BB**	**50% Bb**	**25% bb**	
	Phenotype	**black black**	**black**	**black**	**white**	
5.	**Genotype**	**Bb x bb**	**= 50% Bb**	**50% bb**		
	Phenotype	**black white**	**Black**	**white**		
6.	**Genotype**	**bb x bb**	**= 100% bb**			
	Phenotype	**white white**	**white**			

If you are just about to re-erect that barrier on this subject — don't! . . . Keep plodding on. It really is all quite easy and it just needs thinking about.

If these genes did not separate to go into a new being in re-arranged form we'd all be clones of one parent or the other. Your daughter might have liked to be a clone of Mum, but I don't think she'd care to be a clone of Dad, at least not in structure if he happens to be 6'9 tall and 2 yards wide!

Joking apart, you can see why division is necessary to create a new and different being.

The main point here is that we need to understand how that division and rearranging occurs, as shown in Fig. G.

Say an animal is carrying BB (pure black) it passes down one B to one offspring and one B to another. **NOT** two B's to one offspring. In order to do this the two genes must separate (meiosis) into some other safe corner (gamete). When fertilisation takes place these are received in a pair again.

In Fig. D (page 32) this segregation and coming together created new beings. These had one gene for red and one for white colour inherited from each parent. Depending on the genotype of the parents, the new being can vary, because it can receive this rearranging of genes for two colours in the six different ways described above.

Genetically it can be like either parent — or unlike either parent — depending on the genes for those colours it received, AND HOW THEY WERE RECEIVED. Phenotype of course, it can only be black or white — or red or white (according to the examples shown so far in this book).

Have we answered the question we began with?

DOES LIKE BREED LIKE?

We have to remind ourselves again that the unit of breeding is the entire animal and not the individual gene.

So of course like *may* breed like in some circumstances – especially when you remember that there are many more factors involved with inheritance than simple dominant and recessive characters. But it is highly unlikely in the light of the above segregation.

If that animal does not carry homozygous genes for its physical characteristics it is not possible for it to pass them on – so while an offspring may be *similar* in outward appearance I would not personally be depending too much on like breeding like – in particular where one does not know the history of the animals involved.

By this time any reader who has stayed with it should be able to understand the very simple Mendelian dominant and recessive factors.

Unfortunately not all inherited characters are so easy to understand – nor that simple to explain.

Learning the Mendelian law of segregation first seems to help in getting the more complicated issues to fall into place.

I can now press on to sex determination. I am aware that some might call it putting the cart before the horse, but I really do believe that it is easier to explain at this point – and certainly easier to learn.

TEN

SEX DETERMINATION – X & Y

Remember the experienced farmer (in Chapter 1) who believed our twin heifers to be infertile? – there are many such myths which came from the past and are still believed by some today.

Many experienced cattle breeders believed they could control sex by arranging the time of service of the female.

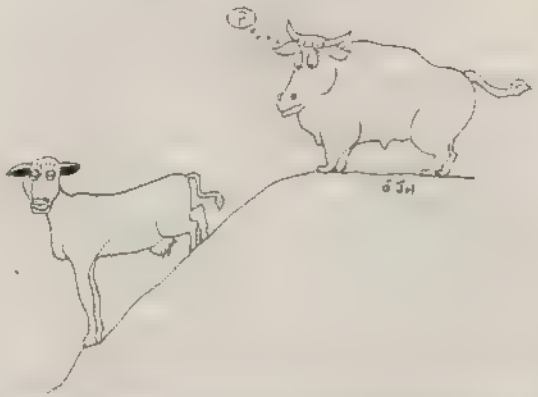
The theory was based on the idea that the left ovary sets free only male producing eggs and the right ovary female. Since the ovaries set free an egg alternatively every three weeks, if they mated at the correct alternate heat they could produce females. This theory is not supported by fact. Obviously any theory can be right some of the time, but the facts are that even with one ovary removed the cow will still give male and female calves.

Based on the same theory as above, another old myth was to put the female on her side. The idea being that if she is lying on her side the opposite ovary is supposedly blocked to any sperm.

This one leaves me with an image of the hefty farmer's wife straddled across the heifer in this gymnastic feat of holding her down – rodeo style! While farmer poses the question of how he will teach his 1000 kg plus bull this un-natural position of bovine love making . . . crazy isn't it?

Another myth is to stand the female on a slope. No one seems to be quite sure if the slope should be up or down . . . or which end of the female should be up or down! It makes little difference to my husband and me . . . our land is completely flat! Learning basic

genetics has not yet shown us a way to breed cattle with tiny short legs at one end or the other — front or back!



NATURE HAS ITS OWN WAY OF DETERMINING SEX . .

It has to do with the X and Y chromosomes.

A CHROMOSOME is a little structure inside a cell on which the genes are carried. In other words, it is a gene carrying body — and we'll come back to this later.

In dogs, cats, pigs, goats, cattle, humans, etc. — in fact in almost all mammals, the sex of the young is determined by the male. (In birds the male has no control over sex of the young).

Today most of us already know this and many of us know how sex is determined by the X and Y chromosomes, but some breeders may not yet have got around to learning this, so I will briefly explain.

Females carry two X chromosomes
Males carry X and Y
} Birds are the opposite

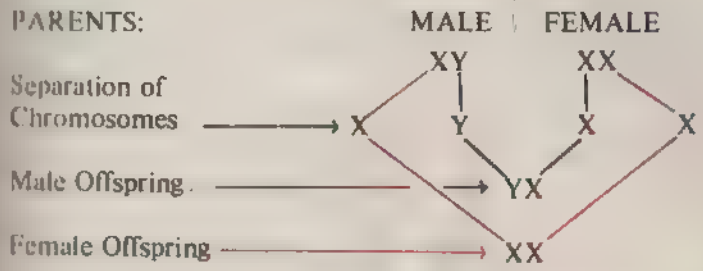
Remembering that the female gives one of what she has to every offspring — and the male also gives one of what he has, it is obvious that:—

The female inherited two X and is said to be XX.
The male inherited X and Y so is said to be XY.

Remember that the female can only pass down what she has — and she has only X to pass. The male has X and Y to pass.

The female passes X to ALL the offspring — the male pases X to his daughters and Y to his sons, as in the following Fig. H.

Fig. H



The y is very much smaller than the X and it has been said that the y seems to be of lesser importance than the X. The X is constantly shuffled back and forth from once sex to the other and according to some text books is clearly not differentiated as female or male.

There is no doubt that there are other sex determining influences at some stage or another, (or under some condition or another) and this entire subject is fascinating, but can get too involved for our purposes here.

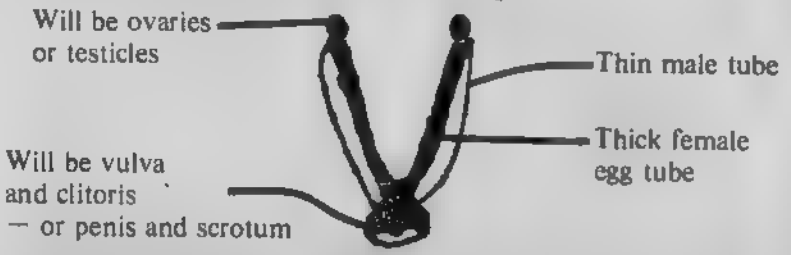
Interesting though at this point, is the case of the true hermaphrodite condition — a person or animal with sex organs of both sexes. More research on this condition seems to have gone into poultry than anything else, and quite clearly it is a common condition in this species. Inter-sex goats it seems are also quite common. So are inter-sex cats. Having been involved with pigs I can assure the reader that this condition is quite common in this species, and seemingly often where intensive inbreeding takes place.

Many years ago we bought a piggery . . . . including the existing stock. It wasn't until we discarded the previous owners' extremely inbred boars and introduced new breeding material that the degree of such abnormalities markedly decreased. This strongly indicated to us that over intensive inbreeding, in particular on unknown genotype, had largely contributed to the increase of abnormalities.

It is possible for a person, or an animal, etc. to have an ovary on one side and a testicle on the other — others can have intermediary conditions. But it is mentioned here purely for interest and further study can be done if the reader so desires. But when we consider how the reproductive organs begin to form, the wonder is not that the odd malformation happens, but that so many creatures are normal. All relevant parts initially exist — to be determined male or female by the chromosomes and chemical action.

Shown below in very crude diagram form, are the reproductive organs after conception. Similar drawings are frequently used in other text books . . . the drawings are easy to understand, so I have merely modified them for simplicity, and added explanations which will make your study easier.

Fig. I – at conception



Chemical action then begins. For the male this means that the thick tube shrivels and disappears and the testicles and penis are formed, as in the following Fig. J.

Fig. J – male

MALE

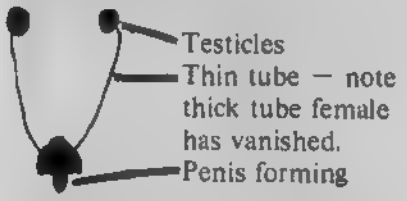
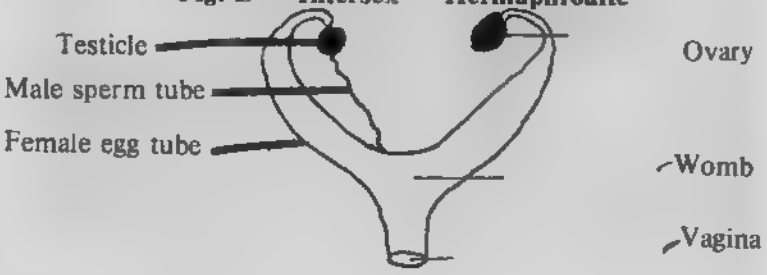


Fig. K – female

For a female the thick tube grows thicker. The ovaries form (in the same place as the testicles shown above). The thick join at the base becomes the uterus, while the lower section forms the vulva and clitoris – in the same place as the male penis above.

It is not surprising then that sometimes things go wrong with the normal distinction of male and female. When it does, like the hermaphrodite, the result may be female on one side and male on the other – drawn again in very rough fashion.

Fig. L – Intersex – Hermaphrodite



When homosexuality is discussed we have all heard it said that animals show no such abnormality. Those who believe this have obviously never worked closely with animals, nor closely observed their sexual behaviour. Animals in fact do demonstrate inter sex behaviour.

Our aim here is to remain with the simple . . . and these conditions are above and beyond the original X and Y inheritance. But the above explanations serve to give you some understanding of why the mode of inheritance is rarely all that simple.

In this book we have to get back to the X and Y.

It has been said that sex is determined not so much by the presence of Y in the male as by the absence of double X . . . sorry guys!

Logically either the Y determines that a particular offspring will be male, or the absence of X determines it.

It is interesting to note that some authorities believe that way back there in history cattle lost the Y chromosome. My enquiries reveal that this suggestion is one that many other experts do not agree with. But those who do favour the theory say that females carry XX and males a single X with no Y. Males are then determined by the absence of the second X . . . which would support the "ABSENT" theory.

In any case the male, and the laws of genetics, sit in the driver's seat – so the farmer's wife can get back to her own chores – and farmer can safely leave his animals to make love in the good old fashioned way they know best.

ELEVEN

SEX LINKAGE

This entire chapter is designed to make the reader buckle down and apply what has already been learned from previous chapters. It will also advance your study into the "Mendelian mode of Inheritance".

Study into sex linkage is not as complicated as it might appear at first glance. In fact it is quite easy, and if you have fully understood the first ten chapters you should have no trouble at all.

In animal breeding very little can really be taken at face value. Learning one thing means you are only one step more towards further study. The mechanism of inheritance is indeed very involved.

Therefore, at this point I have to tell you that genetics in depth is for the GENETICISTS . . . and it is from these qualified people you should seek advice on more involved issues. This sounds a bit like the television adverts for pain killers, which advise that if pain persists you should seek medical advice. Nevertheless it is sound advice.

As I said earlier, we are mere breeders trying to grasp some useful basics for our own interests and improvements — or perhaps with the intention of further study once we've found an easier route into the maze of scientific jargon and complicated texts.

The mode of sex determination is sometimes a bit more involved than a mere explanation of the X and Y chromosomes — as covered in the previous chapter! But in that chapter the existence of the X and Y chromosomes was described, so it should now be much easier to understand why some traits are termed "sex linked".

SEX LINKED simply means a circumstance linked to one sex or another and passed down via the X or Y sex chromosome . . . but most frequently via the X.

In banter I previously deflated the male ego a bit by indicating to them that their little Y might not be all that important. Now I have also to tell them that the X they carry is sometimes the reason why they suffer from problems which are female sex linked. An example of this is haemophilia.

I think we are all aware of this bleeding problem. It is a hereditary defect which prevents blood from clotting . . . and is passed to the affected male via the X chromosome from the female.

For an example of sex linked *defects* the problem of haemophilia is generally used. This is inherited in animals in the same way as it is in man — therefore it becomes easier to explain — and to learn about. Learning the theory involved in this aids our comprehension of many other aspects of inheritance — which we can benefit from!

Haemophilia is inherited in the simple dominant and recessive manner which the reader has studied in previous chapters, except that it is sex linked — carried and passed down through female relations.

Haemophilia is said to be a recessive and is carried on the X chromosome. Most sex linked conditions are carried on the X — and in haemophilia it is considered that the Y is not involved.

What we are concerned with in this book is the *mode of inheritance* of this genetical defect. We need to understand the fundamentals of sex linkage before we can comprehend the more involved issues of any particular defect inherited in this manner.

In the previous chapter it was established that the female carries two X chromosomes, one received from the mother and the other from the father = XX.

The male carries X (which he received from the mother) and Y from his father, = XY

In order to fully comprehend sex linkage it is very important to have studied the previous chapter carefully, and to keep the above two paragraphs in mind. For ease of reference, keep also in mind that the male carries ONLY ONE X which he passes to ALL his daughters. His Y is passed to ALL his sons . . . and in our haemophilia example the Y is not involved.

Just another "reminder". CAPITALS for DOMINANT and lower case for recessive. The DOMINANT in the case is H and it will mask the h recessive. If you did not learn this properly from previous chapters you should now go back and do so.

The key to the abbreviations used in the matings below is:—

HH
XX = X chromosomes with H DOMINANT (normal female)

Hh
XX = X chromosome with h recessive (carrier female)

h
XY = Bleeder male

H
XY = Normal (unaffected) male.

1. The usual mating of haemophilia free stock.

H	**H**		**H**	
X	**X**	**to**	**X**	**Y**

Resulting in all NORMAL (unaffected stock)

2. The mating of carrier female to normal male.

H	**h**		**H**	
X	**X**	**to**	**X**	**Y**

Resulting in:—

	H	**H**
1 normal female	**X**	**X**
	H	**h**
1 carrier female	**X**	**X**
	H	
1 normal male	**X**	**Y**
	h	
1 bleeder male	**X**	**Y**

Half the offspring carry the bleeder gene. This means that half of the daughters are carriers. Half of the sons are bleeders . . . because as seen above, they have only one **X**. On this one they either carry the **H** dominant — in which case they are free of the problem — or the **h** recessive, in which case they are affected. In other words the bleeder has no DOMINANT **H** to dominate over the recessive. The daughters do have another **X** on which to carry the dominant gene — which masks the recessive, so they are impure carriers.

3. The mating of normal female to bleeder male.

H	**H**		**h**	
X	**X**		**X**	**Y**

Resulting in **ALL** daughters carriers. **ALL** sons normal.

This is because the male gives his only **X** to **ALL** his daughters — on which he is carrying the recessive. Why are his sons normal? . . oh! Come on now, you've read it umpteen times! . . . because he

doesn't give **X** to his sons — he gives **Y** to his sons, and **Y** is not involved.

Perhaps the following diagram will help.

Fig. M

	CARRIER MOTHER		**NORMAL FATHER**	
	H h		**H**	
	X X		**X Y**	
GAMETES	**H (X)**	**h (X)**	**H (X)**	**(Y)**
OFFSPRING	**HH XX**	**Hh XX**	**H XY**	**h XY**
	1.	**2.**	**3.**	**4.**
	Normal female	**Carrier female**	**Normal male**	**Bleeder male**

Connecting lines are usually drawn through such diagrams — these can be very confusing.

If you follow the letters down you will quite clearly see that:—

1. Received XH from Dad and XH from Mum = normal
2. Received XH from Dad and Xh from Mum = carrier
3. Received XH from Mum and Y from Dad = normal
4. Received Xh from Mum and Y from Dad = Bleeder

Shown in most text books as:—

1. above XHXH Normal female
2. above XHXh Carrier female
3. above XHY Normal male
4. above XhY Bleeder male

Remember what I said about coining your own abbreviations in Chapter Seven? This type of Haemophilia is termed Haemophilia 'A', so it is often shown in text books as:—

1. above XAXA
2. above XAXa
3. above XAY
4. above XaY

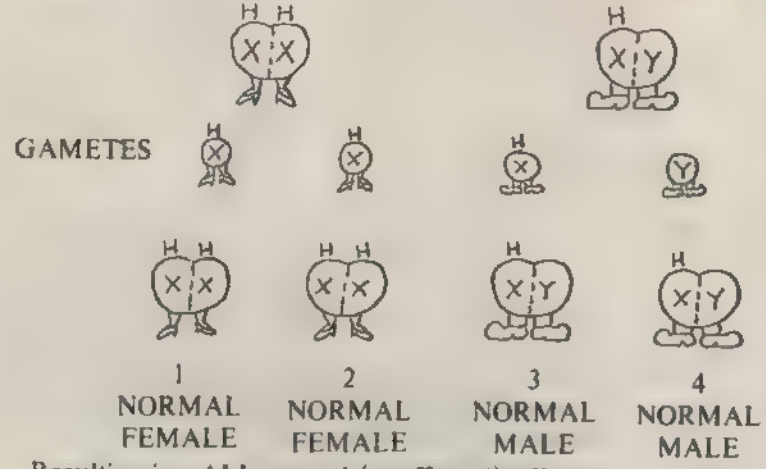
Selection was discussed in Chapter Eight. The difference in selection here is that we are not dealing with mere conformation faults, nor with flaws which might be easily rectified.

We are facing serious degenerate hereditary diseases. Haemophilia is potentially lethal for half the male offspring. No responsible breeding establishment would wish to blindly follow such ill-fated course.

It is said that "one picture is worth a thousand words". To be sure you have grasped the mode of inheritance of a sex linked defect such as haemophilia, (and for the fun of it) let's save 4000 words and have a look at four pictures which tell the story.

(a) USUAL MATING OF UNAFFECTED STOCK

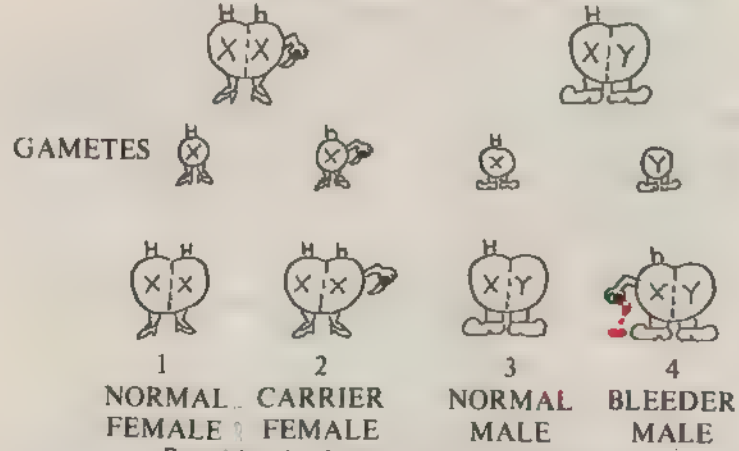
NORMAL FEMALE NORMAL MALE



Resulting in : ALL normal (unaffected) offspring.

(b) THE MATING OF CARRIER FEMALE TO NORMAL MALE

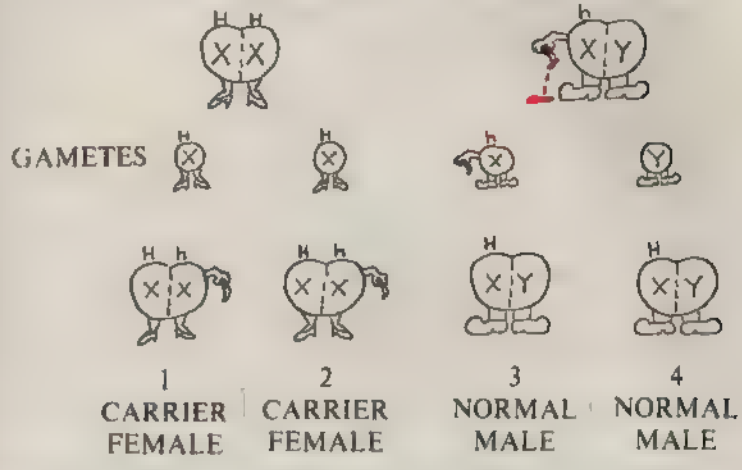
CARRIER FEMALE NORMAL MALE



Resulting in: Half Affected Offspring.
= 1 normal female. 1 carrier female. 1 normal male. 1 bleeder male.

(c) THE MATING OF NORMAL FEMALE TO BLEEDER MALE

NORMAL FEMALE BLEEDER MALE

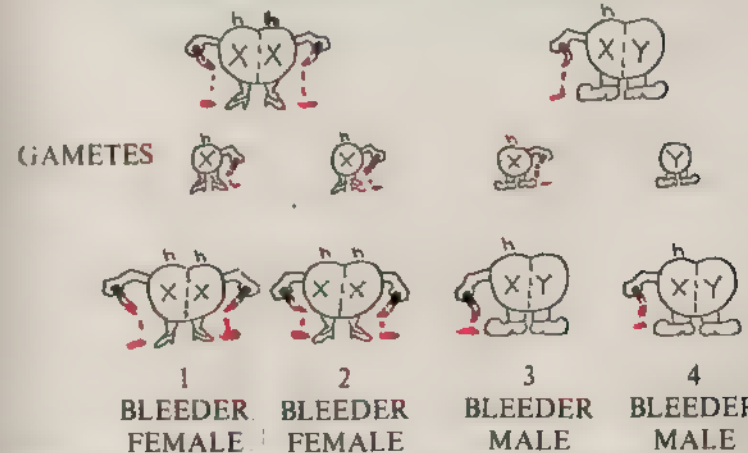


Resulting in: ALL daughters carriers. ALL sons normal, because male passes X to his daughters – Y to his sons.

(d) THE MATING OF BLEEDER FEMALE TO BLEEDER MALE

A very rare mating

BLEEDER FEMALE BLEEDER MALE



Resulting in: ALL offspring bleeders, because ALL X chromosomes (from both parents) carry the recessive h

There are many such hereditary problems which breeders would wish to avoid if possible. Amongst these are jaw deformities, tail abnormalities, epilepsy, hip dysplasia, cryptorchidism (undescended testicles) — just to mention a few. Many of which are not inherited in the above straightforward manner.

However, if we are aware of these things, and learn what we can about their mode of inheritance we are in a much better position to make wiser decisions on future breeding.

Or alternatively what it would be foolhardy to tamper with.

Testing for haemophilia affected males, prior to stud work, is already available for all animal species prone to this problem.

Tests are also underway which will determine the female carriers from the normal — which means that in the future normal females will be identifiable for breeding . . . but until these tests are more readily available country-wide, or if you have not made use of such tests, you live in a fool's paradise if you use females from known carriers.

In fact tampering with any possible haemophiliac stock, without prior knowledge of its mode of inheritance would be like playing Russian Roulette.

Incidentally this sex linkage does not have to be an unwanted factor. For example the poultry farmer is able to use this linkage to determine the sex of his chicks at birth . . . . . in some crosses of poultry the male progeny are all the colour of the mother — and vice versa. This early recognition of sexes proves very economical for such poultry farmer.

TWELVE

INCOMPLETE DOMINANCE

So far we have dealt only with simple dominant/recessive Inheritance.

My attempt to simplify the basics of genetics for you would be inadequate without mention of IMCOMPLETE DOMINANCE.

Many existing text books skip so briefly over this that an attempt to learn from it would be over scant. Other more involved books can be just that — over complicated for most *MERE* breeders . . . . that's us!

While my approach may be a bit unorthodox I would like to explain how I look at incomplete dominance and how I remember the genetics of it.

We all know what a partial eclipse of the moon is . . we know that it is only partially covered. We know that this coverage can be a little, a lot and all degrees in between. At other times there is a full eclipse when the moon is *totally* hidden.

A full eclipse of the moon is complete — the moon is out of sight. Like our recessive when the dominant is present and masks it.

This total coverage of the moon might be likened to the complete dominance which previous chapters have explained. We know the moon is still there (hiding) and will be seen again. We have also learned that the recessive character is also hiding and given the right circumstances will be seen again.

In previous examples the dominance of one of a gene pair over another was complete. No effect of the recessive gene could be seen in the impure (heterozygous) animals. These animals could only show the recessive they carried by further breeding. i.e. the Bb — the Rr etc.

To refresh our memory. If the dominants black or red are present the offspring are black or red. This dominance over the

recessive is termed *complete*. If the dominant is present we see only the dominant character. The recessive cannot be seen, except in double (homozygous) state . . . as in the white pea.

This is not always so. There is what is termed:

INCOMPLETE DOMINANCE

Incomplete dominance may be likened to the partial eclipse of the moon. It only partly covers — resulting in some degree of recessive being visible where we can see it for what it is.

This partial dominance can be a little, or a lot. It only partly hides the partner. This might also be likened to pulling a blind down which has a hole in it — the window is covered but the blue of the sky, or the white of a cloud can still be seen through the hole.

This can be quite easily understood. It is of benefit to breeders because the animal, in some form or other shows genotype as well as phenotype.

When the genotype shows up in the phenotype in this manner, further test breeding is unnecessary.

Remember we had to further breed our impure Bb or Rr to permit us to re-assess and sort out the cream from the poor again? This was because the heterozygote (the impure) resembled only its dominant parent.

Incomplete dominance is different. The dominant only partially hides the recessive allowing some of the recessive to show through . . . . therefore we can see the unwanted character in the living animal, and further breeding to prove the animal is un-necessary.

For example, say black is dominant to all other colours in an animal, if this was incomplete dominance some white would show through . . . telling us, that the white was there in the ancestry.

This white might be a few white hairs scattered throughout the coat — or even patches of white. Or white down the back, up the legs, on the shoulders etc. etc. . . or even a mixture of black and white.

At sale time this display of colours can tell buyers a lot about the ancestry of that animal — which I'll come back to later.

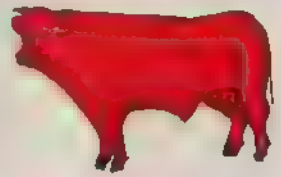
It might be necessary here to remind the reader that this book is not about dogs, cats, pigs, cattle etc. as such. It is about basic genetics — which applies to all animals, fish, etc. . . . and every other living thing. So if you breed cats it applies. If you breed dogs it applies . . . in fact whatever you breed these basic genetics apply.

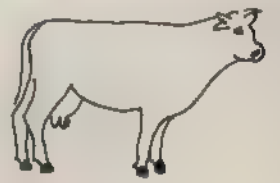
A classic example of incomplete dominance is the mating of red and white in Shorthorn cattle. Using this same example we can easily compare this with our red and white peas — or black and white dogs etc.

In this case red is not completely dominant to white, and the cross results in a mingling of the two colours — a *MIXTURE* of red and white which is termed "roan". So the offspring is physically unlike either parent. The white spreads into the animal's red colour and its phenotype tells us its genotype. The phenotype visibly shows it to be an impure carrier.

Fig. N

RED SHORTHORN **WHITE SHORTHORN**





ROAN

(mixture of red and white)

When these roans are mated together they will *NOT* breed true. They will not breed pure roans. The ratio of inheritance returns to the simple Mendelian ratios of 1 : 2 : 1. Which we have already gone through in previous chapters.

Pure red Impure roans Pure white

The difference is that the roan's phenotype shows us they are carriers of red and white.

As we studied previously, the red will breed true and the white will breed true — the roans will not! It would be just as impossible to breed a pure race of roan Shorthorn cattle as it would be to breed a strain of red garden peas from two recessive whites — only for different reasons as explained below.

Roans bred together produce the ratio of one pure red, two impure roans, one pure white. The roan put back to the white will breed impure roan and white. The roan put back to the red will breed impure roan and red. The roans always remaining impure.

There is also the case of pink flowers. In carnations, for example, the union of red and white produces pink. This is incomplete dominance. The offspring unlike either parent, but a blending of the two colours.

The union of the two pinks will again bring out the ratio of 1 : 2 : 1 = 1 red, 2 pink, 1 white.

The two pinks are showing in phenotype that they are carriers of both. The pink will not breed true and will remain impure . . . as with the roan Shorthorns!

At this point it is interesting to compare the six possible variations from the matings of the above mentioned Shorthorns and the carnations, with the six variations in Chapter Nine of the simple Mendelian black and white. A reader who has applied diligence to the previous chapters should be able to figure out these variations without my help — but we'll do it anyway.

			Producing
1. WHITE	to WHITE	WW to WW	All white
2. RED	to RED	RR to RR	All red
3. WHITE	to RED	WW to RR	Roan
4. ROAN	to ROAN	RW to RW	25% white 50% roan 25% red
5. WHITE	to ROAN	WW to RW	50% white 50% roan
6. RED	to ROAN	RR to RW	50% red 50% roan

I have my own little clue to all this which enables me to work out these ratios at a glance — without getting myself all hot and bothered and out of my depth.

(a) If there are FOUR of one letter ALL offspring have to be whatever that letter stands for, i.e. four WW's have to be white! . . four RR's have to be red.

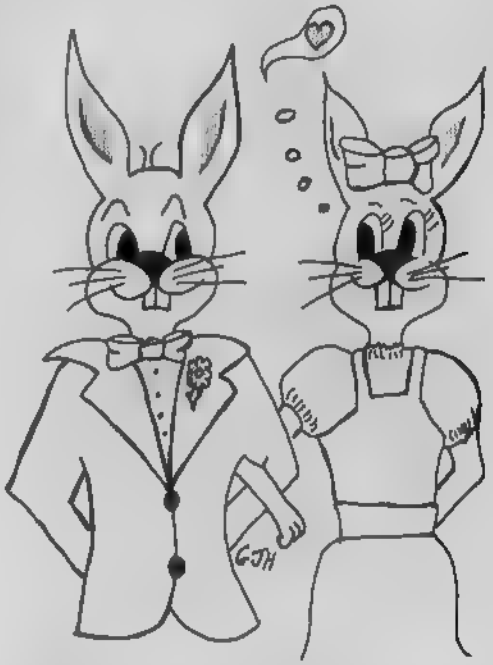
(b) If there are two of each, i.e. WW and RR two of the same on each side — All offspring have to be impure.

(c) If there are two of each . . . i.e. RW ad RW with one of each on both sides, the ratio of 25% pure dominant 50% impure and 25% pure recessive will emerge.

(d) If there are three of one letter and only one of the other, i.e. WW and RW 50% are one colour and 50% the other . . . . but of these 50% must be the colour of the three letters . . . in this case white! Therefore RR and RW 50% would be red!

But back to our incomplete dominance. What we might mistakenly think here is that if all red and white Shorthorns, and red and white carnations were to become extinct there would be no more roan Shorthorns. Also no more pink carnations . . . and one

facetiously wonders how this would affect the sales of the white sports coat the pink carnation adorns.



With modern technology, pink carnations are seemingly unlikely to disappear. A great deal is done with tissue culture and I'm told that x-rays are used to cause mutation for the manipulation of colours . . . . in fact the year 2000 is likely to see a whole new range of colours in many varieties of flowers. Which is all very interesting . . . but will not alter our basic learning of the genetics involved. We have to first learn the basics before we can understand much beyond that.

The basic genetics in this case determines that neither pink carnations nor roan Shorthorns are in danger of extinction for the very simple reason that they carry genes for both red and white — which continue to segregate into that ratio of 1 : 2 : 1, one red, two impure, and one white.

In short, while there are red and white carnations we will always have pinks — and while we have pink carnations we will always have red and whites . . . . and so it goes on without end.

It is the differences associated with the mode of inheritance which breeders generally can use to advantage. The fundamental

difference is that the examples of impure roans and pink carnations are produced by incomplete dominance. The examples of red and white peas (shown as hearts) are complete dominance — simple Mendelian dominant/recessive factors.

In Hereford cattle the white face is dominant to plain face. The offspring of a Hereford crossed with anything else will have a white face. If the cross is with black Angus, black being the dominant to red makes the calf black — but it will have the dominant white face. The same applies to a cross between Hereford and Fresian. This is complete dominance.

If for various reasons this dominance was incomplete — say the Hereford was not pure in the first place, then the face is likely to show irregular patches of the other colour present.

All this is very useful to cattle buyers. When viewing cattle for butchering, fattening or breeding etc. the colour and/or the extent of spread of colour guides a buyer to assessment of what that animals value is for his purposes. For example, if the buyer is seeking calves for fattening for later beef sale he may not be very interested in buying from milking stock unless those calves showed distinct signs of beef parentage . . in one way or another!

Incomplete dominance can show the heritage in the living animal and permit it to be recognised for whatever it is.

Also such buyer can distinguish the crossbred from the purebred by the markings which incomplete dominance manifests.

Some understanding of the traits which incomplete dominance manifests is of equal value to dog and cat breeders etc. as it is to cattle breeders.

The lady who recently brought her Himalayan (colour point) kitten to show me, would not have paid a pet shop an exorbitant price for a supposedly pure bred if she'd taken more time to investigate. The kitten showed distinct markings of a cross bred. She wanted it as foundation breeding stock, so she is in big trouble before she even begins.

Perhaps this brief insight into incomplete dominance will enthuse you to enquire about these aspects of your breeding . . . or enable you to think more seriously about the mode of inheritance of particular conditions which may arise.

With the aid of this book, hopefully you will be able to seek out the credits and debits, the ins and outs, and every hole and corner *BEFORE* launching into a serious, long term breeding project.

THIRTEEN

GENETICAL/CONGENITAL — The Difference

A brief explanation of the terms CONGENITAL and GENETICAL may be useful to the beginner. These terms do tend to become misunderstood.

As breeders we often experience a phone call saying "The pup I bought from you has a problem and it comes from the mother because the vet says it is genetical" . . . when in fact the veterinarian has said it is *congenital.*

There is a difference between these two terms. And yet there can be an association so close that in some instances it may be difficult to separate one from the other.

Nevertheless, there are many factors known to be congenital and not actually genetical. These known ones are the ones our veterinarians generally advise us are congenital — if not understood then it is wise to ask at that visit for clarification from the vet involved.

The Odhams dictionary defines the word congenital as "since birth" or "present from birth". At the risk of being considered audacious I would think a more accurate description would be "present at birth".

Life begins in the uterus. It is here that an animal begins life as a separate individual. All kinds of environmental effects can happen in there. For example drugs are well known to have adverse effects on the unborn. Troubles picked up in the blood stream can reach the unborn and affect its well being. The way a mother is fed has some effect, one way or another, on the unborn.

It is said that the uterine environment cannot form new characters. But it can alter the effects of those characters originally formed.

For example, a high immune system can be inherited in the genes. The lack of immunity our new disease AIDS causes has not been formed in the genes. If the baby was born with it, the defect came via the blood stream of the mother prior to birth, – and in adults via outside influences. Therefore, a high immunity system may have been inherited, but was overcome by environmental influences.

Sometimes external influences affect the full potential of inherited characters from forming. For example we may inherit genes for good, strong bone structure. Yet poor diet while in the uterus, or after birth can produce malformed bone conditions. Rickets is the classic example of a congenital bone condition – formed either prior to birth, or after birth . . . and caused by poor nutrition and in particular to the lack of vitamin D.

At this point I would like to take you back to the twin heifers mentioned in Chapter 1 and 10. To recap on that – for a twin calf to be infertile one must be unlike sexed . . . i.e. one male and one female. The male then takes all the breeding genes from the female . . . well, this is one way of describing it.

Perhaps now we can look a bit deeper into what really happens to that sterile twin heifer our neighbouring farmer thought he knew all about . . . . . incidentally the infertile heifer is called a *FREE-MARTIN*. Its condition is CONGENITAL – i.e. created in the womb after conception and prior to birth. In pigs the same condition is called *WILGILS*. It is sterile, and in some instances partly converted to the hermaphrodite condition discussed in Chapter 10.

Let's be sure we understand what we are discussing. Identical twins are ALWAYS the same sex and ALWAYS from a single fertilised egg. Fraternal twins develop from two separate eggs.

It is these two separate eggs which we are discussing. To get their blood supply these two eggs have to attach themselves to the wall of the uterus. USUALLY THEY BECOME ATTACHED AT DIFFERENT POINTS, and develop quite separately from each other . . . each with its own blood supply.

In the free-martin the eggs may have attached themselves so close together on the wall of the uterus that they are likely to share the same blood supply.

Fig. O

Usual attachment

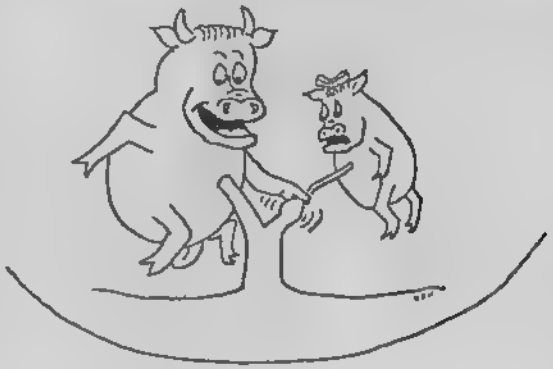
Fig. P

Very close attachment

If the attachment is so close that they share the same blood supply, blood from the bull calf flows through the female and carries with it the chemicals which have triggered off the male characteristics.

This causes the female calf to develop male characterics and by the time of birth the female is neither one sex nor the other. It appears externally to be a female, but internally the vital reproductive parts are not properly developed.

The cartoon below is a friend's idea of the offspring attached too close together on the wall of the uterus, and sharing the same blood supply . . a man's world?



The above described condition would be termed congenital – everything developed normally until by chance the two individual cells became almost as one on the uterus wall.

Having explained all this, there is often no distinct demarcation line as such, between congenital and genetical. Cancer for example is often caused by external influences – but it is now considered there may be some susceptibility towards it in the genetical inheritance.

Another example of environment intermingling with inheritance, is in hip dysplasia in large breeds of dogs. It has been well established that this disease is inherited, and is polygenic, (affected by many genes and not a simple dominant/recessive inheritance, which is fully explained in Chapter 15 on Body Cells).

The *DEGREE* by which the animal can be affected by hip dysplasia may be changed by external influences. This can include such things as overweight in youngsters, incorrect feeding and incorrect exercise.

Having written that I'm also very much aware that this excuse cannot be used as a reason to shun breeder responsibility in this regard, because without doubt it is first and foremost a hereditary problem.

Nevertheless, outside influences can create a higher grade severity of an inherited problem in excess of the initially inherited degree.

THEREFORE, BECAUSE WE NOW HAVE A HIGHER GRADE OF PROBLEM EXAGGERATED BY EXTERNAL FACTORS, WE CAN NO LONGER ESTIMATE THE DEGREE OF THE ORIGINAL INHERITANCE.

We have established that each animal has lived in two kinds of environment. One in the uterus. One after birth. And prior to these its genetical inheritance was determined.

For the novice, simple descriptions which avoid involved confusion and are relatively safe, are:—

CONGENITAL — present at birth, but not necessarily inherited.

GENETICAL — inherited (or suspected as being so).

Even though these may be somewhat loose definitions, a beginner will not go far wrong by remembering them in this easy way.

Having understood something of the difference between these two words means that you have guidelines to follow when deciding whether to retain, or discard, parent stock which have already given you birth defects.

FOURTEEN

CHROMOSOMES — Gene Carrying Bodies

I have previously mentioned chromosomes. For a better understanding we now have to look further into these gene carrying bodies.

Genes have been likened to beads on a string. Unfasten the clip on the ends of a string of beads — hold one end in your hand and let the rest hang down and you have something similar to a chromosome with its genes attached.

The string is the chromosome — the beads are the genes.

You will make the following study easier if you beg, borrow or buy some 4 ply wool. Preferably white — or a lightish colour. 4 ply string will also serve the purpose.

Having acquired your wool then draw a circle on paper. This circle is our pretend cell.

Cut off a piece of 4 ply wool — about one to two inches. Split the wool into two separate pieces. This will now be two lengths each with two strands of wool . . . or string.

Put your two lengths inside your circle. You now have two *PAIRS* of chromosomes in your cell.

Fig. Q

Toss out your existing pieces and cut another length from the 4 ply. This time use a red pen to put a mark at the top of the wool being sure to have some red on all four strands.

Do the same with a green pen in the centre of your length . . . then do the same again with a black pen to make a black mark at the bottom of the length.

Now split it in half as before. There should be some red on the top, some green in the centre and some black at the bottom . . . ON EACH HALF! And it is important to be sure the colours have penetrated all four strands.

Place your two 2 ply lengths again into the cell (circle).

And hey presto! you now have two pairs of chromosomes in a cell, with your three colours which represent genes on identical positions on the chromosome pair.

Fig. R

The genes (red, green and black) have established their appointed home on the chromosome. Their location! Their LOCUS!

LOCUS (plural LOCI) is latin for *PLACE*. It means a seat — a place in which something is situated.
For our purposes here it means the appointed place for genes on the chromosome. Their location.

To return to our Fig. R. No matter how you shuffle these pairs of chromosomes around those genes (colours) will stay on their own locus. This is the place you appointed for them with your coloured pens, making this the home for them. You can put red to red. Black to black. Green to green. All like to like and homozygous. Or you can have red to black — unalike and heterozygous.

If you split both these lengths again — into one ply you will have four strands, on which the genes still stay on their appointed place.

These four separate strands would then not be termed chromosomes. *Chromosomes occur in pairs* — so half a chromosome after separating, (i.e. a single chromosome) is called a

CHROMATID. What you have now is four chromatids . . . or you can call them two pairs! It is important to understand this for further study.

Cut off another length from your main supply and repeat the whole procedure to Fig. R. Only this time use different colours for the second piece. You will now have six colours to experiment with.

Split these as previously explained and you now have four *PAIRS* of chromosomes . . . all with the genes on the appointed place . . . some coming together with matching colours (homozygous) and others not matching (heterozygous).

This is an elementary idea of how chromosome pairs split (separate) into two halves and in doing so each half keeps the relevant genes on their own locus.

Now wasn't that easy?

Later we will go further into this chromosome separation. For now, try to remember that during the separating process the half chromosome (one strand) is called a chromatid, on which is carried a identical half of the genes from the original chromosome pair.

Play around with your four pairs of chromosomes. Having split and separated your wool you can now have:—

Red to red.	Green to green.
Black to black.	Red to black.

plus the extra combinations of the new colours you introduced.

While experimenting with this, add a few more lengths and a few more colours.

Satisfy yourself that you have understood the above fully.

Then separate all the lengths you have into single strands (chromatids). Mix them all up together in one heap, and begin extracting them out one at a time — i.e. at random, and pairing them up again.

When you separate your strands note that because you dictated where the genes would be, that is where they always stay, on their own location on that chromatid.

And take particular note of the fact that when you pair them together again, pure chance dictates which colour they meet up with.

If you add even more colours and more lengths you can then see how complex it all becomes, and how the chance of one of a pair of genes finding its partner can be a very random affair.

You must then wonder if it is all so uncertain how can we put such knowledge to use? Knowing that some will match up — and

how they match up, gives a breeder more opportunity to increase the homozygous genes by planning with genotype in mind, as well as phenotype.

The most important part of the above experiment is that it makes it perfectly clear that if you have not used a colour, say purple in your above experiment then it is impossible for purple to be present in your final result.

Nor can you get a desired improvement in progeny if the parents do not carry favourable genes for it.

Therefore, what is carried on the genes manifests itself in the living animal . . . but remember it is not necessarily showing! and sometimes only further breeding will bring it to light.

The clue is really in the fact that when a fault does come to light in recessive (pure — homozygous) form then you are quite sure that both parents carried it — after studying the above you'll have no qualms in discarding it for further breeding. That is one very big step you've already made towards improvement.

Here I have to go back to the dominant/recessive study we've already been through. If your red above meets red you have purity for red. If red meets black you have impurity for either colour. If green meets green you have purity for green . . . and so on!

What you don't know is which is dominant and which recessive.

Assuming the green is a recessive fault, then green meeting green will bring its effect out where you can see it. . . . and you know that this is homozygous (pure) for your undesirable feature!

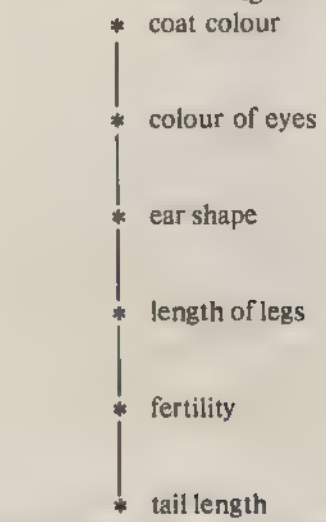
Assuming that red is responsible for red colour. Black is for black colour. Green for erect ears. Blue for short coat. And considering all four to be dominant. Our little effort with the 4 ply wool also enables us to see more clearly how the dominant/recessive operate.

Fig. S

Dominant			Recessive
Red colour	■	□	White colour
Erect ears	■	□	Floppy ears
Black colour	■	□	White colour
Short coat	■	□	Long coat

Another way to show you how your 4 ply wool and colours helped your understanding of genes is this imaginary strand of six genes showing their function.

Fig. T



This study will also help us to understand how outcrossing, linebreeding and inbreeding affects the gene locations on the chromosome. These breeding systems are fully explained in Chapter 18. For now, we can define them as (a) inbreeding — breeding closely related animals together; (b) Linebreeding — mating somewhat related animals together, and (c) outcrossing — the mating of unrelated parents.

Obviously outcrossing generally *RESHUFFLES* the genes in quite disorderly fashion.

Line breeding has a better chance of matching them up — but not necessarily matching up the desirables (you have to take the bad with the good).

Inbreeding does an even better job of getting the homozygous genes together — but the breeder using this method would need to be very well informed on the material he/she is working with. The chances of disastrous results is very much increased.

I make no comments on the merits of these three systems of breeding, because I think this is a personal choice which breeders make for themselves. The purpose of this book is to supply you with knowledge which may help when you have to make this decision.

History has shown excellent specimens of all breeds, of many species, from all three systems.

Now we know how genes are shuffled back and forth as the chromosomes separate. In other words, how genes get separated and

come together again. And it is essential to understand that some get detached in the process. . .

. . . . remember some will match colours, while others will be mismatched. In addition to this, not all chromosomes are the same length, so some genes get left without partners.

If you refer back to your wool, when the lengths were extracted from the pile of single strands some would be much longer than others and pair up with shorter lengths. This would leave some colours stranded on the ends. In future generations these may again meet a partner — or they may remain isolated for many generations — but they are never really lost! More on this in Chapter 17.

What we still have to learn is how the chromosomes get separated in the first place.

So we must move on to cells and cell division.

FIFTEEN

BODY CELLS

The genes are carried on the chromosomes, and the chromosomes are located in the cells of the body.

The amount of chromosomes carried in each cell depends on the species involved.

For example man is known to have 46. The dog is said to have 78. Cattle and goats are believed to have 60. Cats 38. Rabbits 44 . . . and so on!

Remember we learned with our 4 ply wool that chromosomes are carried in *PAIRS* so these numbers have to be halved — i.e. man has 23 *PAIRS*. Dogs 39 pairs. Cattle 30 pairs and cats 19 pairs.

I have explained that these chromosomes are carried in the cells of the body.

There are two kinds of cells.

1. Body cells. 2. Sex cells.

BODY CELLS. These divide and multiply in a way which ensures that every cell and its chromosomes are *EXACT* duplicates of each other. All body cells have the same content. These cells ensure that if you breed sheep, you get the structure and all the things which go into forming a sheep. . . the head, the eyes, ears, legs, brain etc. of a sheep and not some other species.

SEX CELLS. These divide and multiply in quite a different way — which will be explained later. For now we can say that sex cells are mainly responsible for various changes from generation to generation, within a species.

For simplicity, the difference lies mainly in the way the chromosomes divide and come together again.

This can all become a very complicated study and is obviously not easy to simplify without involved explanations.

So in order to minimise confusion between the two I will keep these two studies apart from now on.

The body cells will be dealt with in this chapter and the sex cell in the next one.

The simplest way to study body cells is to begin at the very beginning of life itself.

The female sheds an egg. It is fertilised by the male sperm. A new being is born. This new being's body is made up of thousands of cells, all of which contains the correct number of chromosomes for that species.

To make our study simple let us assume that the being we have just created has a diploid (total) number of four chromosomes (two pairs) in every cell in its body.

Remember the 4 ply wool? All the above means is that you are back to the start of that chapter. Previously you split the wool in half. And you put the two (two ply) pieces into one cell.

Now you can make TWO circles (two cells). You will split the 4 ply into two 2 ply pieces. You will then separate one of your two ply pieces and you will put half into one cell and the remaining half into the other.

In other words, one chromatid in one cell and its partner in the other.

Then do the same with the second piece. Now you have exact duplicates of the wool in both cells.

For the moment forget about the colours. What we are dealing with here is the cell and chromosome division.

The chromosomes keep splitting down the centre and going half into each cell — and the cells keep multiplying until cell division is completed within the body . . . and each cell has received the correct number of identical pairs of chromosomes for that species.

I think by now you will have understood the chromosome division in the body cell, but to be sure of a better understanding of the cell division it is necessary to explain further.

It is important to realise from the above, that if you put all your wool into one cell there would be far too many chromosomes in that cell for that species.

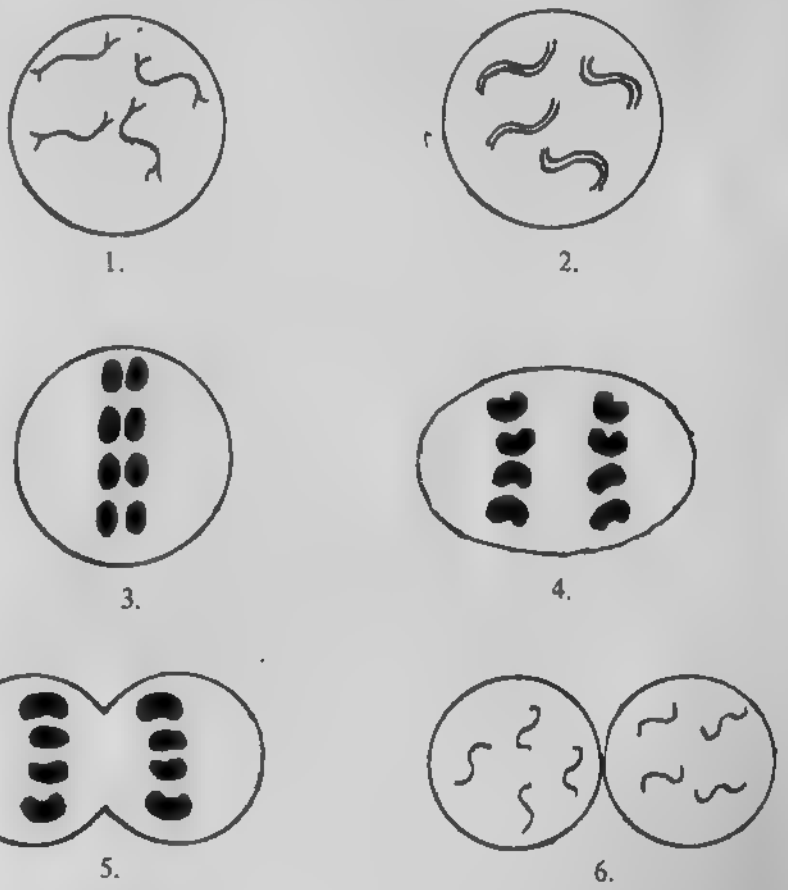
So the cell itself has to divide — taking half the chromosomes with it. It divides into two, then four, then sixteen and so on. When this process takes place each cell then again has its correct complement of pairs.

Below is how the cell divides — remember that the chromosomes split down the centre and separate.

How they separate will be clearly seen in the following diagrams. These drawings are a little over-simplified in comparison with similar presentations in many other text books. My reason for doing this is to simplify your initial understanding of the process involved, so that you can progress into those more involved text books.

The first cell has begun with two pairs of chromosomes. The correct amount for our pretend species.

Fig. U 1 to 6.



1. The cell containing two pairs of chromosomes. Note they are already beginning to split down the centre, to form two halves.
2. Chromosomes separated. Now four pairs. Diploid number now eight.
3. The chromosomes thickening and now arranging themselves ready to be drawn towards the outer edges of the cell.
4. The chromosomes being drawn to outer edges of cell. This is described in text books as polarisation. I imagine it as a magnet pulling the chromosomes to opposite sides of the cell.
5. Cell beginning to divide in the centre. This might be likened to a balloon which you have squeezed in the centre.
   Keep squeezing the balloon in the centre until these two cells divide — each half taking with it two pairs of chromosomes. Four diploid.
6. This now becomes two cells each with the two pairs of chromosomes we began with. Which are the correct amount for our above pretend species.

In this same manner these two cells keep dividing. They make four — then eight — then sixteen and so on until the body has its full requirement of body cells.

Each time the cell divides it takes with it the correct number of chromosomes for that species, whether it is four, or sixty . . . and so life goes on!

If you wish to look up, or study further into this duplicating and dividing of cells. The text books sometimes call them "ordinary cells". They are also termed "somatic" cells. The process of division is called "mitosis".

In the text books you will also come across "polygenic" inheritance. Big word — again! But when you remember that *poly* merely means MANY it then becomes easy to see that the word "polygenic" simply means many genes involved . . . many factors! However, while it is quite easy to remember the word, it is not easy at all to understand what is really involved in terms of inheritance.

This is not a simple inheritance as was our Mendelian mode of inheritance. For example, the hip dysplasia in large breeds of dogs, which was previously discussed, as a complex polygenic inheritance. This cannot be predicted in terms of an expected re-appearance of the problem in the F2's. Therefore, it becomes much more difficult to reduce the incidence of it — within one's own breeding establishment, and throughout a breed.

When breeding time comes around, the patterns of polygenic characters inherited from the body cells are much more difficult to alter. An unwanted problem inherited in a polygenic manner often

requires the discarding of severely affected stock, and the use of animals showing the lowest **DEGREE** of an abnormality.

No simple rules can be given for the reduction of polygenic abnormalities. However, it is a safe rule never to breed from any animal showing an abnormality which affects the well being, or function of that breed.

On the other hand, it is also very important to remember that in withholding an animal from breeding because it may transmit one unfortunate defect, all its good qualities are also sacrificed. Such qualities might, for example, be rare in that breed. In which case a breeder is then faced with the question of what takes priority (discussed in Chapter 19).

This becomes very much easier to appreciate when you realise that the body cells, chromosomes and genes on them multiply, then divide and separate in exact duplications of each other.

This study makes an enormous difference to your breeding plans.

For instance, if a problem is inherited in a simple Mendelian factor, given time and a knowledge of the mode of inheritance you can reduce the incidence. And you may be able to achieve your improvements from the stock you have already.

Whereas certain polygenic problems, not being so easy to follow the inheritance pattern of, sometimes requires the elimination of parent stock.

Remember the old adage that a stable is only as good as its brood mare? Well, now you know why!

But then you may ask, "If duplication is so exact why is it that every individual is different in looks, character, etc?" (except in the case of identical twins.)

Of course it is not all as clear cut as that — what we've learned are very elementary beginnings. We must now go on to learn a bit more about sex cells, because these are mainly responsible for making each individual different.

SIXTEEN

SEX CELLS

The main concern for breeders regarding sex cells is in the effects it has on the animals we breed rather than (or as well as) the manner of inheritance.

Because the sex cell chromosomes separate in a different way from the body cell chromosomes explained in the previous chapter, they also have different effects.

It is this variation of effects which is so valuable to breeders.

Sex cells separate in a way which ensures that each individual born is remodelled into a new and different being.

This explains why brothers and sisters — human or animal — all have a somewhat different physical appearance even though they may be strikingly similar to each other, or to the parents. They have also ■ different genetical inheritance . . . which we have already learned is more likely to show up in the second (F2) generation.

The method of separation is one aspect of inheritance which is most important to animal breeders. If you by pass it you miss out on knowledge which is invaluable.

When I began this book I promised simplicity — even if it sometimes meant being a little unorthodox (and laborious) in order to get the basics across.

So I'll simplify this in a way which I personally used to grasp the beginnings. Once I'd done it in the way shown below I found I could then go back to the intricate text books and absorb it in more detail. I hope it will do the same for you.

We have actually done a similar exercise before. You might like to go back to chapter nine and revise on the first introduction of the words *gamete* and *meiosis.*

MEIOSIS explains the dividing of sex cells. It is involved with segregation — the separating and coming together again of sex cells.
Remember that when the cells divide, the chromosomes and genes split up and divide with them.

My dictionary of Biology uses one and a half pages (approx. 700 words) to define meiosis. This should make you aware that the brief explanation above is for our (breeders) purposes, and is consequently minimal.

GAMETES are half cells formed from the meiosis division. In the process of meiosis the easy way to remember GAMETES is to think of them as the safe corners for the chromatids and genes to take refuge in until fertilisation takes place. See Fig. G, and Fig. V.

In the body cells we have already learned that the chromosomes split exactly in half, taking the genes on their own loci with them. Each half of the chromosome together with its genes is an exact duplication of the other half. This division then goes on until the developing embryo has its full requirement of body cells.

An embryo is an offspring in the process of developing in the uterus. Birth of a species is the expected end result of an embryo.

Simplified we can say that one of those cells developed in the embryo will be a sex cell. Without sex cells the species cannot reproduce.

In the body cell of the animal each chromosome with its genes on their own loci, are exact duplicates of each other. This is not so with the sex cells.

When the sex cells (sperm and egg) are formed only ONE chromatid goes into the sex cell — so the sex cell in the first place gets only half the genes compared to the body cell which has a full compliment of genes.

Therefore, when the sperm meets the egg and fertilizes it, the offspring gets half its genes from the male and the other half from the female parent.

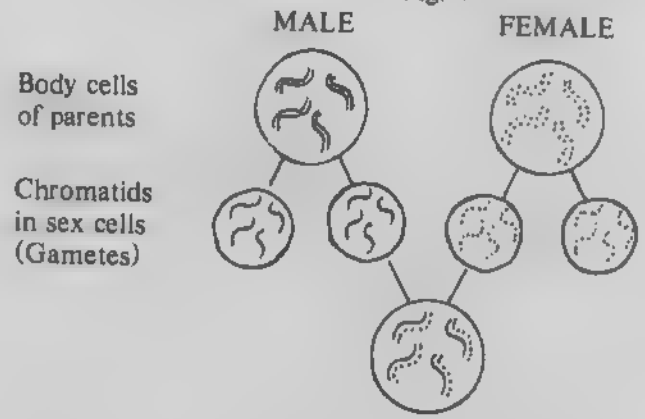
And much more importantly these genes are carried on only half a chromosome pair — a chromatid from each parent . . . ensuring that the offspring receives a completely different assortment of genes when the two different chromatids joined up again at fertilisation.

This can best be shown in diagram form because the jargon involved can bewilder we mere learners. As explained previously, once you know the initial principle a more intricate study into existing text books can be within your grasp.

Let's assume that the full compliment of chromosomes for our species is 4 pairs. The male has four pairs — the female also has four pairs. Somehow we have to fertilize these without giving the new offspring eight pairs in each cell. Eight pairs for this assumed species would be abnormal so nature must reduce the number so that each offspring gets its correct four pairs.

We are going to mate these two together and see what happens to the chromosomes in division. The drawing 'V' below, is just another version of similar, commonly used diagrams in other books. In this instance, I decided to stay with this familiar presentation because it is already simplified.

Fig. V



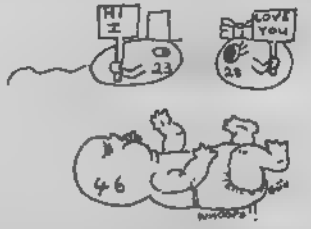
Body Cell of new individual

From this it can be seen that the chromosomes are still splitting down the middle to divide. But instead of being drawn to each side of the cell, and separating balloon fashion as the body cell did, these chromosomes actually part company. Each half then singly goes into its own safe corner (gamete). At fertilization they come together. One from the male the other from the female parent — resulting in the correct amount of chromosomes in the *FERTILISED cell, for that particular species. In this way the* sex cell ensures that a different individual is formed.

Let's re-cap on that. The embryo began as one *FERTILISED cell — described above! From time of conception its BODY* cells then began the process of division by duplicating that exact same cell over and over again until the *BODY* had its full compliment for that species.

Say, for example that species was man, who has 46 chromosomes in each cell. The body is made up of multiples of these

exact same cells . . . except one! which is a sex cell. This contains only half that number of chromosomes — i.e. 23. This sex cell at time of mating meets the sex cell of the mate and with a "Hi! I love 'yer" they unite and form another being with the correct number of 46.



It is important to remember though that they come together again at random. It is pure chance which chromatids meet, and the more chromosomes for the species the more variation there will be. This also means a random allocation of genes.

Each individual offspring receives chromosomes of different type from each parent. Which in turn ensures each individual a different genetical make up from its parents — or from its relatives (except identical twins).

This ability to inherit varied, or changed characters permits a breeder to use the knowledge to improve selection — and it allows scope to breed on again from those improvements.

It also means that while the male often boasts the credit for all improved offspring, the female in fact is of considerable importance to improved breeding . . . and from the above you can see why! — she contributes half of the full compliment of chromosomes, with her half contribution of genes on them. So she is going to influence the characters the offspring inherit, just as much as the male does. . . . whether it is for good, or bad!

In other words say the male has the magnificent head you wish to reproduce, and the female the poor head you wish to avoid. You might expect to get some (or all) good heads in the offspring — but you should also be aware that you might also get some (or all) poor heads from the female.

What we have to be mindful of in improved breeding is that in using the better head over the poor head the resultant offspring, if not showing the better head, may indeed be carrying the required genes to give it. If this is so, it may not show up again until the F2 (second generation).

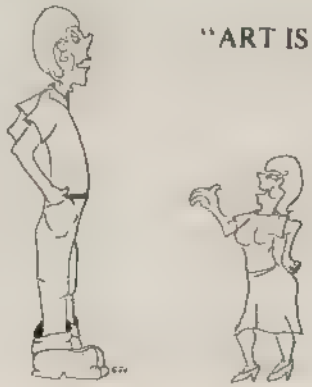
Very often the first generation is discarded by the breeder before the desired characteristic bred for has a chance to emerge in the second generation.

Of course you can't have the good without the bad and the same applies to the poor head. It may not show up in the F1 (first) generation but has a fair chance of showing up again eventually — most likely in the F2 generation.

This is only one example of where understanding the basic principles of the division of chromosomes in the sex cells can be of enormous use in a long term breeding establishment.

* * * * * *

NOTE: The above explanations are how scientists to date have believed chromosomes are arranged at random in the sex cells.
The Plant Breeding Institute in England have recently claimed a new discovery. Their research is said to be showing the chromosomes may be arranged in patterns, rather than at random.
These patterns, if they have their facts right, may change all previous understanding of the arrangement of chromosomes in the sex cells.
The scientists involved explain that with the modern technological methods available to them, the chromosomes are distinctly showing as a three dimensional pattern with the chromosomes from one parent arranged in a concentric pattern around the chromosomes from the other parent . . . . and that genes probably determine how these will be spaced in the nucleus.
This all very interesting — and very exciting! But at the time of writing this book, I can only explain these things as they are understood at the moment.
Genetics is the newest science. Obviously there is plenty of scope for more enlightenment into existing beliefs and for new discoveries.
To keep up, even in a very elementary fashion, my reader needs to look into these aspects from time to time, and this simple book will give the reader the basics to be able to do that.



"ART IS LONG — LIFE IS SHORT"

SEVENTEEN

CHROMOSOME — CROSSING OVER

Genes have been likened to beads on a string. They have a home on the chromosome — a locus! We have seen this for ourselves in our exercise with wool and coloured pens.

This exercise was simple. It made it seem as if the genes stuck to their loci no matter what — in a sense they do. However, other things can happen which disrupt their effect.

Chromosomes can do strange things. During the dividing and coming together again they can break. In fact they may not all be of equal length in the first place. If you go back to your lengths of wool and begin cutting them up into unequal lengths and then try matching your colours up, you will see that sometimes you leave colours on their own on the longer lengths.

Now you have some sections long while the others are short.

Common sense says that the longer the chromosome the more room there is for genes. The shorter the chromosome the less room.

For example you might cut off the blue on the lower end of a piece. What you then have left is its partner blue stranded on the longer length.

Fig. W

The gene on the longer length might then be separated from any possible partner, as in blue above. If it doesn't find a partner, it will remain dormant.

What is likely to happen is that it will pair up again with another blue in subsequent generations. You may not have known you had a blue in either parent and when this blue pops out again you wonder where could it have come from?

This is a very good reason why records should be kept on ancestors and offspring. The only way you can know the material you work with is to keep every bit of information you can gather — right from the very first litter you produce. Follow through as much as

possible on everything you breed. Also check out as many progeny as possible from any litter mates of your own adult breeding stock, and store as much information on the ancestors as you can acquire.

In this way you may not be quite so surprised when something odd occurs — even better, you may avoid a mating which could produce the oddity. Of course the trait may not be undesirable — in which case you will be clapping your hands and working at reproducing it.

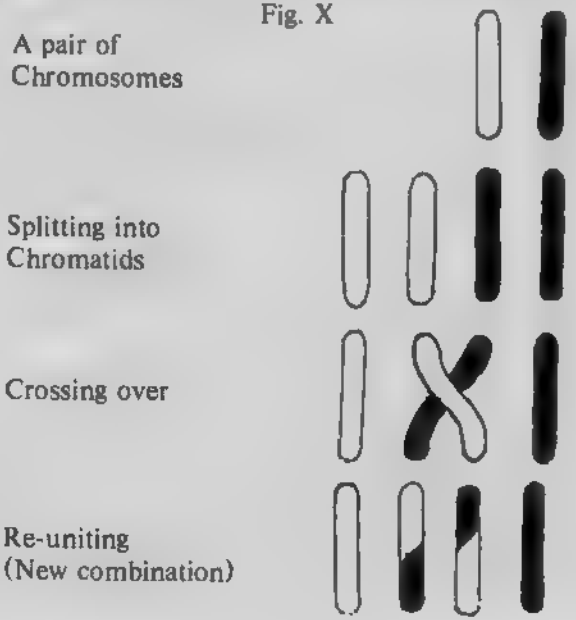
Another thing which may happen to the chromosomes is what is termed "crossing over". This process is an important one because it contributes to making sure each individual is different.

Chromosomes splitting down the centre were shown in Chapter 14, using 2 or 4 ply wool to explain. Sometimes the chromosomes do not make a clean break down the centre at time of division. In other words they may not separate down the original juncture. Part of one half might cling to the other half.

An animal *MUST* transmit its correct amount of chromosomes for that species. However, it can transmit these in a crossed over manner so that part of one is mixed up with part of the other.

This crossing over can cause one part of a chromosome to become fused with another part. Fig. X. below gives a rudimentary idea of how crossing over can occur. Similar simplified diagrams are shown in most text books.

Fig. X



This can mean that genes which were partners and otherwise might have united together to form a character, are now re-arranged. This, to the breeder can mean that some character you hoped to get was lost in this shuffling of genes. It can also mean that new characters emerge — which again may be desirable, or undesirable.

It also means that an animal may receive chromosomes which are in fact not half from the male and half from the female as previously described. If the chromosome crosses over the resulting pair may be two-thirds from the male and one-third from the female, or vice versa — ah well! genetics wasn't meant to be easy!!

This process, like all genetical processes, can be much more complicated than indicated above. Again breeders need only to be aware of it in order to understand how changes take place. This process contributes towards variation. Variation contributes towards change. Change contributes towards evolution and the continuation of a species which can adapt to its environment.

Without crossing over, and other variations of inheritance, a breeder would have no challenge. It is the possibility of changing for the better which keeps sincere breeders interested, and involved.

This crossing over causes genes which were previously opposite each other — for example on the 4 ply wool — to separate away from the neighbouring partner.

It can occur at any part on the chromosome — adding even further to random selection.

This variation makes our role as breeders less predictable, all that much more challenging, and consequently more exciting.

EIGHTEEN

BREEDING SYSTEMS EXPLAINED

Outcrossing, linebreeding and inbreeding were mentioned in Chapter 14.

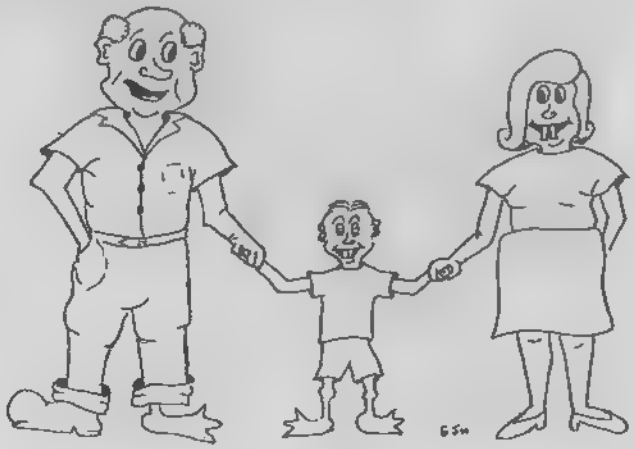
I said I would not comment on the *merits* of these breeding systems because history has shown that excellent animals can be bred from any of them.

We do, however, need to know the meanings of these terms because plenty of poor and mediocre specimens are also bred from any of them.

This is mainly because animals can be *PREPOTENT* for their characteristics — irrespective of whether or not this was planned by us.

Firstly we have to learn the definition of the word prepotent. In this book we are concerned mainly with the correct usage of the term, because the term DOMINANT is often used when PREPOTENT was meant.

PREPOTENCY is the power of one parent or another to transmit its characteristics to offspring.



Say for instance a female constantly passes on an excellent head, this animal is said to be prepotent for this feature. On the other hand she may be prepotent for passing on a poor head. Remember you have to take the debits into account as well as the credits.

DOMINANT was well covered in previous chapters. By now you will be very well acquainted with the usage of this term. For our purposes DOMINANT is probably best reserved as a genetic term, explaining the dominant gene over the recessive.

Whereas prepotency can be remembered as more of a physical effect.

For example, if a mother is prepotent for a particular type of head, she can be seen to give this type of head regularly throughout her offspring — and often irrespective of the mate used on her.

This female can mostly be depended on to give this head and we can see it! So we say she is PREPOTENT for that feature. The term DOMINANT here would be incorrect.

Prepotency to pass on characteristics to offspring (i.e. homozygosity) may be a result of dominancy — but the two terms have different usage.

An aspiring breeder would find that a clear understanding of the usage of *prepotent* and *dominant*, is a great asset in deciding which breeding system to follow.

Also it behoves a breeder to learn what choices of breeding systems there are.

To make this explanation complete all systems are mentioned — these are:—

In-breeding
Line-breeding
Out-breeding
Out-crossing
Back-crossing
Cross-breeding

IN-BREEDING

This is generally used to describe the closest possible relationships. These would be parent to child or brother to sister. In human terms we call this incest. Half brother to half sister would fall also into this category.

The aim of in-breeding is to increase homozygosity (purity) for as many desirable features as possible. Logically the debits also have to be considered. Pitifully, ■ used by many breeders, in-breeding merely increases homozygosity for poor and mediocre characteristics.

The success, or failure, of in-breeding depends not so much on the system as on the human beings planning such matings.

The very idea that anyone would launch into such close inbreeding without any idea whatsoever of the material they work with, nor any knowledge of basic hereditary principles, would give a more enlightened breeder goose pimples.

Nevertheless if a breeder has the right material in the first place, and does have a knowledge of basic hereditary principles, the advantages of this system are that type is fixed more quickly than usual and the prepotency of the animals involved is increased (again for good and/or bad). Heavy culling (discarding undesirables) goes hand in hand with this system.

Therefore, in my opinion in-breeding would *NOT* be an easy launching pad for beginners to dabble in.

LINE-BREEDING

This system seems to be the more popular method amongst breeders. It has many of the advantages of in-breeding and somewhat fewer risks. But risks still remain.

Matings are still made to related animals, but to animals not so closely related. For example Uncle to Neice — grandfather to granddaughter. Or great-grandfather to great-granddaughter etc.

Strictly speaking this system is mating to a particular ancestor . . . i.e. forming a line of descent from a particular ancestor.

What applies to in-breeding also applies to line-breeding. Knowledge of the stock, and the ancestors, together with an understanding of basic hereditary principles is just ■ essential. To line-breed or in-breed to mediocre animals is likely to produce only more mediocre stock and unlikely to produce anything in the way of excellence.

In both in-breeding and line-breeding the quality of parent stock is the first essential.

OUT-BREEDING

This term is not in common usage because it is similar, in principle, to *out-crossing* which is a more commonly used term.

It is merely the opposite to in-breeding and means mating to lesser related, or unrelated animals. An example might by type to type mating with little, or no relationships involved. Logically it may also mean re-shuffling the previous gene combinations into a heterozygous (impure) arrangement.

It can be used to advantage when new features are required, which cannot be bred from existing stock.

Its disadvantage is that it increases heterozygosity (impurity) — which in turn decreases prepotency.

It may result in an inheritance of good and bad . . . . in which case logic would indicate that an F2 generation needs to be planned in order to take advantage of the laws of segregation. Putting this to practical use in this way would also obviously include strict culling procedures — possibly for many generations to come.

OUT-CROSSING

Everything that has been said above of out-breeding can also be said of out-crossing.

Strictly speaking the method is (or should be) mainly used for the introduction of new genes for characters which do not exist in a currently running inbreeding, or line breeding project.

Genes which do not exist in the first place cannot be inherited. Therefore sometimes it may be necessary to outcross away from an in-breeding or line-breeding project in order to acquire a desired character.

The experts advise a return to line-breeding after the outcross is made.

One of the main disadvantages of such outcrosses is that undesirable genes may also be inherited along with the favourable ones hoped for.

Therefore, it is generally recommended that out-crosses be made away from a line-bred strain, only when absolutely essential. Even then a breeder is advised to choose out-crosses from other line-bred strains in order to see in the phenotype of the offspring what that animal's genetical make up produces . . . i.e. do the out-cross mating between two line bred animals, (each having proved their prepotency for the characteristics desired).

BACK-CROSSING

This system merely means mating back to one ancestor or another . . . i.e. child to parent.

The system is far too often used as an excuse for incest . . . . the breeder having dabbled in a child to parent — or brother to sister mating often feels the action has to be excused by saying it was a test mating. When asked what they were test mating for they invariably have no answer.

If you feel you have enough knowledge to do a back-cross, or an incest mating you should know why you are doing it and what you hope to achieve by it — there should be no need to seek excuses for doing it!

For test mating, which brings recessives to light, it is better to stay with the term "test mating". Such tests are usually made to identify carriers of degenerate problems, in order to eliminate them

from a breeding project. Or for research workers seeking information.

While the term "back-crossing" is obviously closely associated with the terms "test mating" and "in-breeding" there is that subtle difference which can be confusing for us if we don't have some way in which to define these terms.

The term back-crossing is easier for a beginner to clarify if used to explain the mating of the heterozygote (the hybrid) back to one of its homozygous parents . . . i.e. the F1 back to the P1 — after which the laws of segregation will separate the F2 generation into the ratio of 50% PURE, 50% impure and so make future selection a little easier. At this point if you are not sure about this segregation percentage, go back and recap on Chapter 9 — in particular No. 2 (Bb to BB) and No. 5 (Bb to bb) of the six mating variations.

The term back-cross is best understood as a mating back into parent stock.

CROSS-BREEDING

This is the term for the mating of two pure bred animals of different breeds . . . i.e. a pure bred Hereford to a pure bred Fresian — or a pure bred Labrador to a pure bred Cattle Dog.

As I said before, it is not for me to recommend, or decry any of the above systems. The choice is a personal one and must be made by the individual breeder.

I have included these explanations to enable you to think about them, and to encourage you to weigh up the advantages and disadvantages for yourself.

NINETEEN

GETTING THE PRIORITIES RIGHT

Knowing which weeds to remove from our gardens means a better showing of all the beautiful flowers and shrubs.

The "eye for ■ weed" has to be acquired.

Let someone loose in the garden, who can't tell the difference between weeds and flowers, and shame on them — what a mess!

It is said that an "eye for a dog" . . or horse, or whatever . . is inborn!

Astute breeders are sometimes called "canny breeders". Meet one! . . . talk to one for any length of time and generally you'll find that they have a lot more going for them than just an "eye" for an animal.

However, I'm not about to question this "eye" because it has some credibility, for it has been said that animal breeding is more art than science.

Nevertheless, a blending of both art and science would seem to be the more efficient approach.

If we are not amongst these "favoured few" who do have this "eye" then we must acquire it.

We cannot clean out the weeds if we can't recognise them.

Animal breeders need to be able to recognise the excellent from the mediocrities. Having the knowledge and ability to do this is a valuable asset. There are no cut and dried guide lines. These have to be learned.

For example you may be faced with the choice of which one of two females to breed from. One is an overall mediocre specimen, while the other is a quality animal with one glaring major fault.

Should you breed from the mediocre animal?

What I would term "mediocre" is a rather poor, below average specimen — neither good, nor bad! Such animal probably has no serious faults, but then also has no special virtues.

This overall mediocrity is, in my opinion, just as serious a defect as any single fault.

With careful breeding a single fault can be got rid of — or reduced in incidence. In particular where such fault is controlled by a simple dominant and recessive mode of inheritance.

Logically, if we have an outstanding animal with say only one fault, (even a serious fault — but obviously not DEGENERATE desease) commonsense breeding can correct it.

To me, an excellent animal with one glaring fault would be a better breeding proposition than the mediocre specimen — provided a breeder realises that selection for correction of the fault is vital from thereon: This could mean quite a few generations of selection before the improvement is "fixed".

Complete mediocrity — with no superiority to offer — is likely to pass on only more complete mediocrity.

Even if the male used excels over the female, and the mediocrity does not totally manifest itself in the F1 generation, some of the offspring will certainly carry the genes for it.

For example, a first class bull over a herd of inferior heifers might show some improvement over the mediocrity in the first generation from him. Even so, every single calf from this herd is carrying the genes of inferiority from the heifers.

It might be said that *ANY* improvement is better than none at all, and of course this is so!

What we are concerned with here are hereditary facts which, by being aware of, you can gain from.

It is also essential to know the difference between major and minor faults.

Without prior knowledge of the range of faults we may be faced with in whatever animal we breed, we cannot benefit from the basic genetics we have learned. If you are unable to recognise a major fault from a minor one, no golden insight into genetics is going to tell you where your priorities lie at selection time.

Having learned the major and minor faults of your breed, you then have to learn to get your priorities right, in relation to the *ENTIRE* animal and its purpose — rather than to one isolated fault.

For example, if you have a "light eyed" animal in an excellent strain of working dog, and the accepted standard of that breed requires "dark eyes", this fault might be frowned on in a show ring, but would not affect its working performance . . . so it would be a minor fault for a working dog.

If the above animal lacked stamina, intelligence or correct

temperament, these faults might be overlooked in a show ring, but would most certainly affect its performance. So they are major faults for a working dog.

Therefore, you could well breed on from the light eyed dog — striving towards a darker eye if you wished. The other "overall unsuitable" dog you would want to discard from future breeding of working dogs.

On the other hand if showing is your main interest in breeding animals, then again you have to get your priorities right. If the fault is frowned on in the show-ring (and even though you know it is only a minor fault) you would then need to take this "light eye" aspect into consideration in future selection.

Getting these major and minor faults into the right perspective is not all as easy ■ it sounds, and yet it is crucial to future breeding plans.

Having got them into perspective they then have to be viewed in relation to their mode of inheritance.

For this we need another example. Say we have a breed which requires erect ears and the dog you hoped to breed from has drop ears. Should you breed from it?

Obviously it would be far better to start with a dog without this problem. But remember seeing it is recognising it.

So, by seeing it you just might be generations ahead of someone else whose dog doesn't show it, but carries the gene for it which will manifest itself in future generations. They have the same problem as you. Only they can't see it — you can!

The best dog to begin with is obviously one which neither shows it nor carries it — but remember we have no way of knowing what the animal carries until it is bred from — except in those instances where the recessive has come to light — as in our dog with drop ears!

In this instance, we previously learned that erect and drop ears are inherited in a simple dominant and recessive manner.

Therefore, it should prove no problem at all to correct such fault within one or two generation — provided you have learned your basics well.

If the fault had been "polygenic" (multiple genes involved) then the entire story changes. We have limited control (if any) over things which are polygenic in nature, and sometimes the only way to get rid of such problem is to discard those animals from breeding . . . i.e. use only animals which are not affected. This decision has to be made according to the severity of the affliction and the effect if has on the animal.

I can only give brief examples, like those above. It is up to you to learn more about your own breed and its requirements . . . if you do not do this, genetics (simplified or not) in the end result will not be of much use to you.

The last chapter includes reading pedigrees to your advantage. You cannot find what you want in a paper pedigree if you do not know what you are looking for in the living animal.

Everything eventually boils down to this vital business of assessing the living animal — its requirements, and your purpose for breeding it.

The showring is often adrift from the working scene — and the commercial field is often remote from both of these.

The common denominator being the desire by all to produce improved specimens. Which is also the hopeful end result of our study here.

TWENTY

STEPPING IN THE RIGHT DIRECTION

To recap, the closer together homologous genes are located on the chromosomes the more likely they are to unite and pass on their qualities — good and bad! Keep this in mind when reading the rest of this chapter.

You will remember that homologous genes are the ones which are alike. Earlier in the book these were the BB (black) and the ww (white). And if black was our desired feature, learning its mode of inheritance would aid in future selection for it.

You know that homologous genes exist — you learned this in previous chapters. Being aware of it and putting it to use are two different things.

By studying this chapter you will be able to use what you have learned. From your pedigrees (plus of course the compatibility of the physical animals) you can adopt a breeding plan which will bring the homologous genes closer together where they are more likely to influence the next generation.

Earlier I discussed the benefit to breeders of constantly breeding towards the cream, and discarding the undesirable.

Before this could be fully understood the basic principles of genetics had to be grasped.

I trust by now you are studying the stock you have and wondering what genetic material you have in those animals of yours. Well, there isn't much point in standing gazing at the animals. We've already seen that phenotype is no certain indication of genotype.

Nevertheless phenotype is the manifestation of the genetical inheritance — so your phenotype will need to be a good type of that species, or breed. So on second thoughts, do go and gaze at your animals, but do it realistically.

For example, if they are show dogs, cats, cattle, pigs, horses etc. Do they meet with the standard of that breed? All show animals have an accepted standard of points which you should aim for.

Do you know that standard? If not, this must surely be top priority for your immediate study.

If you are breeding for commercial purposes do your animals meet with the requirements of that market? Poor animals take just ■ much caring for as excellent specimens. They cost as much to feed, and generally fetch lesser market prices! Associated costs, such as provision of adequate accommodation or veterinarian charges, are not reduced for poorer animals – nor are charges raised for superior animals. Only the initial cost of purchase is higher, so always start with the best you can afford – this is very old but very sound advice.

If you have working animals, are they as bodily sound as they should be? Are they as intelligent as they might be? Is character as reliable as it ought to be? If you breed from unsuitable working stock, then you must expect to breed more of the same.

Other than all this, when breeding time comes along where might you begin to get some sort of idea into the possible genotype?

There is a way which can help considerably. You may get a good guide from the degree of in-breeding (linebreeding) in your pedigrees. This will be the pedigree of the male animal you intend using at stud, as well as the female.

This means that *BOTH* pedigrees must be studied.

It is a good idea to rewrite both pedigrees on to one . . . i.e. make up a pedigree of the expected offspring of the mating. Do this *before* you do the mating.

In this way you can see what animals might appear once, or twice etc. in the pedigree of the progeny.

You are seeking the improvements in the progeny – not in the parents! . . . it's too late to improve the parents!

The degree of line breeding (or inbreeding) will give you some idea of whether or not homologous (akin) genes might be close together where they are most likely to be transmitted to the offspring.

This degree of inbreeding is called "the coefficient of inbreeding". It is too involved to go into in this simple book – but you can get under way by making a pedigree mean much more than the mere mention of a few ancestors.

Simplified, the term coefficient means co-operating – uniting – combining. So for us it means the co-operating, combining or uniting of homozygous genes from both parents to bring about a specific result. This understanding permits us to work out the

coefficient – or the percentage – of inbreeding already done, or about to be done. And from that percentage what results might be reasonably anticipated from future planned matings.

There is a very old breeding formula which supports the following theory:–

"LET THE SIRE OF THE SIRE BE THE GRANDSIRE OF THE DAM ON THE DAM'S SIDE"

A variation of this might be the sire of the sire being the grandsire of the dam on the SIRE'S side.

Such formulas clearly bring homologous genes closer together, but are mentioned here solely to give you incentive to think about them. Remember that your plans need to vary to suit the material *YOU* have, because the first thing you have to do is acknowledge where your own stock fails or excels. Consequently no formula as such can be consistently recommended.

Everything eventually boils down to the phenotype and genotype you begin with – plus the quality which you aim to produce.

For our purposes here it is enough to say that if the animals you intend breeding from have any degree of in/line-breeding at all it is important for you to find out just what they are in/line-bred to.

Say you have one line-bred to "Big Joe". Have you studied Big Joe *AND* his ancestors? Is Big Joe the type you wish to breed? Is Big Joe worthy of line-breeding on? What credits did he have in his favour, and what debits should you be taking into account?

You have also to take into account how long ago it was when Big Joe was alive – was it five years ago? . . ten? . . . twenty? Would he be considered a good specimen *TODAY?* . . . or is he merely the best of what could be bred many years ago . . . and long since superseded?

Merely quoting in-breeding (or line-breeding) to some remote ancestor just because that animal was an award winner, or a notable producer in his day – or because he was bred in some other country, or an imported animal, is not realistic.

To brag that an animal is sister, brother, or some other relation to a superior specimen is also a wee bit silly – and having got this far in this book you will realise how foolish this is for yourself.

To confirm this, all you need to remember is the chapter on sex cells which confirmed that every new being is a unique individual (other than identical twins).

However, if you are going to be duplicating genes which are likely to manifest their characters in the offspring, then you need to satisfy yourself that the animal you are in/line-breeding to is what

you really wish to breed today. And that it may duplicate for you the best of its most likely qualities.

Looking at a pedigree and seeing only big names and show awards might be good for the ego, but for breeding improvements this is a short term attitude.

By all means have your day – even the best of breeders have been there and done that – we are all human! Go ahead and gaze – go ahead and gaze long and lovingly at all those impressive pedigree names, and show awards . . . but come breeding time come down to earth!

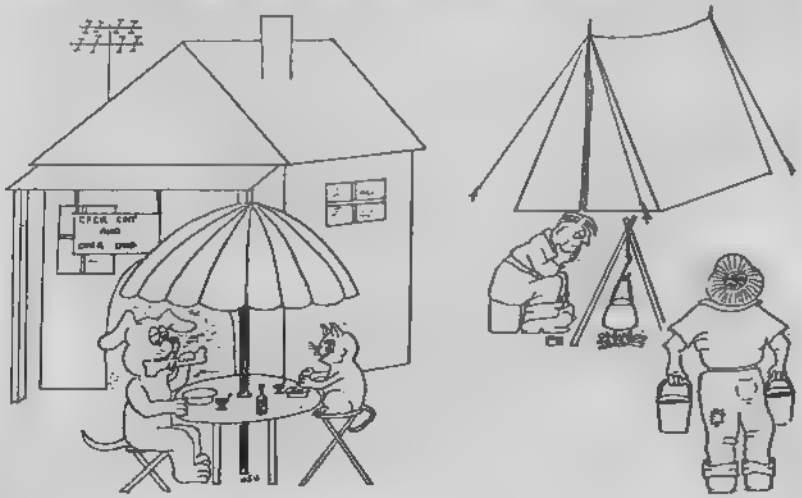
I think one of the most ridiculous sayings I've ever heard is, "My animal has a pedigree as long as your arm" . . . if it has a pedigree at all that pedigree has to be as long as the breed has existed, or as long as that breed has been registered as such. In other words *EVERYONE'S* pedigreed animal will have a pedigree as long as your arm.

A pedigree is merely a list of names – a history of ancestors! . . . not a guarantee of quality! *A PAPER PEDIGREE IS ONLY AS GOOD AS THE ANIMAL IT BELONGS TO.*

Your main concern is whether or not you will be able to reproduce the physical qualities required.

If you do not intend to attempt this, then you are still floundering – relying on chance! . . . go to gaol and begin reading again at Chapter 1.

IF you are serious about breeding improved specimens, and *IF* you can keep your head – because many breeders do seem to lose theirs . . . . then you will need to learn something about the



inheritance possibilities via a pedigree. *THIS IS WHERE YOU CAN PUT YOUR PEDIGREE TO GOOD USE.*

You should have formed some idea of what you expect (or hope to get) from every mating.

The pedigree and perhaps a photograph, may be all you have if, for example, all ancestors are overseas.

Photographs are usually unreliable simply because they can be (and often are) touched up. Which leaves us with the pedigree. Let's have a very brief look at what we might do with a pedigree . . . . bearing in mind the above comment that the first consideration must obviously be the living animals.

Most pedigrees have a minimum of four generations. Altogether there are the names of thirty animals on such pedigree. Two in the immediate parents (P1), four in the P2, eight in the P3 and sixteen in the P4.

Common sense should tell us that although there may be say two famous ancestors amongst those names this still leaves twenty-eight animals which have to be taken into account because they have all contributed to the next generation, in some degree or other.

Two famous ancestors aren't going to contribute any large percentage of influence over those other twenty-eight *UNLESS* the two are one and the same animal (i.e. common ancestors) or alternatively closely related to each other – in which case the percentage of influence increases.

The degree of this influence varies according to how many times a name is duplicated, and how near or far such names are from the expected progeny.

Say for instance that such names are a duplication of the same animal. For example Big Joe in the P1 on the father's side and Big Joe in the P2 on the mothers. Make a note that you have this animal in the P1 on the sire's side – simply put down the name of the dog (Big Joe) and number 1.

Big Joe 1

To distinguish between the sire's side and the dam's side the semi colon is generally used (;). Now what you have to write down is the number 1 to say the animal appears in the 1st generation on the sires side – and a semi colon ; which says you have now finished with the sire's side and are proceeding to the dam's side of the pedigree.

You now have Big Joe 1 ;

We said that same name cropped up again in the P2 of the dam's side – so we now add this number 2 after the semi colon. 1 : 2 and we put it after the name of the duplicated animal.

Big Joe 1 : 2

You can now quite easily see that you have a common ancestor appearing twice in the pedigree – once on the sire's side in the first generation and once in the dam's side in the second. By using this method of reckoning you know all this at one quick glance, and without the above lengthy explanation.

How can you put this to practical use? One of the main uses is that it abbreviates your study and avoids otherwise copious notes and records. If the same animal appeared again in the P4 on the sires side and in the P3 of the dam's side, it would then be written down as:–

Big Joe 1,4 : 2,3

Meaning:– (1,4 on the sire's side and 2,3 on the dam's), and giving you the inbreeding extent of the planned offspring.

If you can learn to read pedigrees in this manner you can progress from there into other text books which teach you how to calculate the percentage of in-breeding *USING THE ABOVE METHOD OF READING THE PEDIGREE.*

Even if you never learn how to calculate the coefficient of inbreeding, the fact that you have an animal duplicated ■■ close to your progeny as 1 : 2 (or 1,4 : 2,3) will strongly indicate that your chances of bringing homologous genes together are quite high.

Knowledge of these things assists you in making decisions on suitable matings.

You can then increase your chances of bringing the favourable homologous genes closer together where they are more likely to influence the next generation you are planning. Also, and just as valuable, this knowledge allows you to avoid **doubling** on animals which are known to pass unwanted traits.

If you have purchased stock from ■ complete outcross mating there will not be two names alike in your pedigree. In this case you are gambling. Where do you plan to go from there? . . . most breeders do not know and when asked this question the answer invariably is "I haven't thought that far ahead yet". I will return to this total outcross later.

Having bought your breeding stock, breeding time is just around the corner – probably less than two years away for your young stock. Two years is a very short time to cram in all that study – so the time to think ahead is now!

Begin by drawing up the pedigrees of potential matings. Take time to fill in as much detail on faults and virtues, as you can gather.

Having done this then extract any duplication of names from it. Then put even more work into research on the duplicated animals, because it is from these you hope to pick up your homologous genes. So , it is wise to know as much about them as possible.

An example of what is meant, and gained by the above might by shown in German Shepherds.

Say a dog is duplicated fairly close up on both sides of the pedigree – say 1:2 inbreeding on this dog. Say he also happens to be the type of overlong bodied – overdeep chested – snipy muzzled – or short legged German Shepherd accepted some years ago, (and incidentally, just now rapidly fading from the Australian scene). To serious breeders such type would be most unacceptable today – either in the show rings, or for breeding stock.

Therefore, no matter how famous the animal was in his day, it would be un-wise to deliberately double up again on this dog in *YOUR* modern project for improved specimens . . . . especially if in phenotype or genotype, the female you intend using carries some, or all of these faults.

Extend this one example to the many other problems which can be associated with our attempts to produce sounder animals – hereditary problems such as epilepsy, dwarfism, haemophilia, cryptorchidism, and hereditary blindness (to mention just a few) and you can see that without the above study your entire efforts will always remain a very hit and miss affair.

We have to return to the animal which is a total outcross, with no linebreeding to grasp on to. All is not lost – everything depends on the breeder's aims.

You could continue to type breed – many superior animals come from this system. However, I have to repeat that your first essential is the standard of that breed. Is either parent a worthy specimen?

Alternatively you might use what you have as a sort of seed bed. You could decide to plant something more superior in this seed bed and proceed from there. If that animal has one near ancestor of excellent type, you could grasp on to this by in-breeding to a worthy close relation and "breed up" from there. Breeding up merely means a slow, planned improvement with each generation – building up a gene bank for higher quality as you go.

Of course this is going to take longer, and may be much more costly in the long run. Also, bear in mind that while you are doing this, there will be other breeders who start better and who may remain ahead of you. Needless to say, a knowledge of genetics is

going to be of considerable use, together with that essential knowledge of the breed.

If you wish to proceed from that animal by breeding better quality for the future as you go – slowly gaining some control over the genotype as well as phenotype, then now is the time to begin learning and planning. Not *AFTER* you have wasted time and money in stocking your establishment with poor quality animals.

Knowing some basic genetics is not enough. And I must stress once again that the genetics you do learn can be put to worthwhile practical use . . . and produce rapid improvements for you, but . . . .

***ONLY* IF YOU INITIALLY LEARN TO RECOGNISE A GOOD SPECIMEN WHEN YOU SEE ONE, BECAUSE THE BREEDING UNIT IS THE ANIMAL – NOT THE GENE!**

The two studies – genotype and phenotype absolutely MUST go hand in hand.

Remember the myths in Chapter 10? Myths – misconceptions – misbeliefs – misunderstandings – mistakes – mismanagement – what's the difference? They all stem from inexperience in one way or another, and most from a lack of learning more about what is inolved before we launch into breeding.

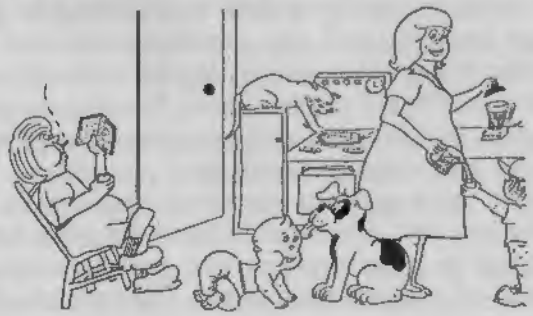
Having worked your way through this book you are to be congratulated, because it was your own wish to learn which got you this far. I hope my presentation of the subject in a simple and lighthearted manner has contributed towards your enthusiasm.

You are now advised to buy, or borrow every other text book you come across because there is still SO MUCH MORE to learn. To urge you on consider the rewards and remember this:–

Dreams alone will not achieve realistic results, and further study requires your time and diligence.

Did I hear you bemoan your lack of time? Now! Now! – no excuses! Remember the proverb . . . .

"IT'S THE BUSIEST PEOPLE WHO FIND THE MOST TIME".



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GLOSSARY

Chapter

ALLELE. One of a pair of genes, situated on the chromosomes, and usually controlling contrasting characters. 9

BACK-CROSSING. Used to describe the mating of a hybrid back to one of its parents. 18

BODY CELL. Cells which are identical throughout the body. 15

CHROMATID. Half chromosome, after the division of a pair. 14

CHROMOSOME. Gene carrying, thread shaped body. q.v.* 14

CROSS-BREEDING. A term generally reserved for the mating of two pure bred animals of different breeds, within a species. 18

CROSSING OVER. The exchange of sections, or parts, of the chromosomes from the male and female. 17

CULLING. The discarding of undesirable animals. 18

DOMINANT. The gene which has the power to manifest its own effect, while inhibiting (masking) the effect of its recessive partner. q.v.* 3

This masking effect may be complete or incomplete. 12

EMBRYO. An offspring in the early stage of development in the uterus. 16

FILIAL. Offspring. Abbreviated to F – i.e. F1 a son or daughter; F2 a grandchild. q.v.* 4

FRATERNAL TWINS. Twins developed from two separate ova, and are not identical. 13

FREEMARTIN. An imperfect female calf, twin with a male and incapable of breeding. 13

GAMETE. The germ, or reproductive cells – but it is easier to simplify them as half cells from the division of sex cells. 16

GENE. The unit of inheritance.
Attached to chromosomes, and passed from parent to offspring. q.v.* 14

GENOTYPE. The hereditary aspects. q.v.*
– in contrast to the phenotype. 2

HAEMOPHILIA. An inherited condition in which the blood fails to clot. 11

HERMAPHRODITE. Unisexual – having the reproductive organs of both sexes in the one animal. Usually sterile. 10

HETEROZYGOUS. Unalike – opposites – impure. q.v.* 4

HOMOZYGOUS. Alike – similar – pure. q.v.* 4

HYBRID. A cross between parents which are genetically unalike. i.e. the first cross between different breeds, or species. 4

IDENTICAL TWINS. Genetically identical. From a single fertilised egg. Of the same sex. 13

IN-BREEDING. Mating of close relatives. 18

LINE-BREEDING. Mating of less related individuals. 18

LOCUS. The home of a gene on its chromosome. 14

MEIOSIS. Involved with the division of sex cells. 16

MENDELISM. Science of the behaviour of genes. 3

MENDEL'S LAW. 1. Of segregation. 4

2. Of Independent assortment. q.v.*

MITOSIS. Involved with the division of body cells. 15

OUTBREEDING. Mating of unrelated animals, or lesser related animals. 18

OUTCROSSING. Mating of lesser related animals 18

P1.2.3 etc. Parental generations. q.v.* 4

PHENOTYPE. Visible manifestation. The animal we see. It is possible to have the same phenotypes with different genotypes q.v.* 2

POLYGENIC. Many genes involved. 15

PROPOTENCY. The power to consistently pass on certain characteristics to the offspring. 18

RECESSIVE. Opposite of dominant. The gene which is masked by a dominant partner. q.v.* 3

SEX-LINKAGE. Sex linked genes usually carried on the X Chromosome. 11

SEX-DETERMINATION. 10

TEST MATING. Mating of a nybrid back to a known recessive. 18

WILGIL. An imperfect condition in pigs, as Freemartin in cattle. 13

* q.v. Referred to in other chapters.